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THE MALARIA PROBLEM IN AUSTRALIA AND THE AUSTRALIAN PACIFIC TERRITORIES.

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INTRODUCTION.

UNTIL malaria assumed a military importance in the Pacific campaigns, the extent of the problem in the Australian region was not generally recognized. The malarious areas under Australian administration were scattered, difficult of access and of wide extent. They supported a sparse white population and possessed only meagre economic resources. Few important centres of population or large industrial ventures existed which could demand the facilities usually required for effective malaria control, or the detailed investigation necessary for its establishment. Consequently, till 1942, when the war reached New Guinea, adequate malaria control measures had been established in few places in the Australian region. Important basic studies had been carried out by a small band of workers, mainly of the staffs of the Australian Institute of Tropical Medicine at Townsville, and of the School of Public Health and Tropical Medicine of the University of Sydney. But facilities for such investigations were not extensive. The greater part of the local pre-war knowledge on the subject appears in the publications of Baldwin (1922, 1930), Breinl (1912, 1915*a*, 1915*b*, 1918), Cilento (1924, 1928, 1930, 1940), Cooling (1924), Cleland (1912, 1915, 1928), Edwards (1924), Heydon (1923*a*, 1923*b*, 1927), Hill (1917, 1922, 1925), Holland (1933), Mackerras (1927), Maplestone (1923), and Taylor (1917, 1918, 1927, 1934*a*, 1934*b*).

With the extension of the war to the Melanesian islands Australian troops were placed in highly malarious areas, and it was at once apparent that a system of malaria prevention was necessary which was capable of withstanding the stresses of jungle fighting, and adequate to cope with conditions of hyperendemicity which had invariably proved disastrous in tropical warfare. The prime military importance of the problem resulted in the establishment by the Australian Army of a comprehensive organization for the prevention of malaria, for the large-scale investigation of the problems relating to it, and for the training of every soldier in the methods of personal protection and "Atebrin" suppression. As a result it was found possible, for the first time, to maintain units as effective fighting bodies in hyperendemic areas, practically without mortality from malaria.

In addition to the problems encountered in the highly malarious battle areas, attention was necessary to the clearing of endemic foci which existed in civil communities in north Queensland and Northern Australia, where were situated the important forward mainland bases, and where troops staging, or in transit to Pacific areas, were likely to be infected.

The further problem of the introduction of malaria into the potentially malarious areas of Northern Australia was also important militarily, as well as from the viewpoint of the civilian public health, for malaria could be expected to cause a dangerous wastage in troops in the northern defensive positions. For this reason the situation had to be dealt with promptly, and involved extensive questions of survey, area control and administration, and the rigorous exercise of powers which are available only in such emergency.

The success of the Australian Army measures against malaria was due in the first place to General Sir Thomas Blamey, Commander of the Allied Land Forces in the

South-West Pacific, whose quick understanding of the problems involved, and skill and forcefulness as a soldier made him preeminent as a leader in the tropical campaigns. To the Director-General of Medical Services (Major-General S. R. Burston) and his officers, and especially Brigadier W. P. MacCallum, a debt is owed which can be realized fully only by those who understood the magnitude and importance of the tasks for which they were responsible, and the administrative skill and unsparing labour with which they built and maintained the medical organization upon which rested our successes in the New Guinea campaigns.

Upon the large-scale researches of Brigadier N. Hamilton Fairley, F.R.S., Director of Medicine, and the Land Headquarters Research Unit (under Lieutenant-Colonel R. R. Andrew and later Lieutenant-Colonel C. R. Blackburn) at Cairns was securely based the remarkably effective chemotherapeutic control that formed the main antimalarial measure in the Pacific battle areas. In fixed positions it was possible to limit transmission by vector control, but in the stress and movement of jungle fighting drug suppression was indispensable. From data provided by specially planned experiments it was proved to the General Staff that strict "Atebrin" dosage could control malaria under the most strenuous conditions of service. This was accepted, and formed the basis for the careful training and rigid antimalarial discipline that altered the course of the war in the Pacific. In addition, important studies were made on "Paludrine" and other drugs, and a large body of information was gathered incidentally on entomological and parasitological aspects of the disease (Fairley, 1945*a*, 1945*b*, 1947; Fairley *et alii*, 1946*a*, 1946*b*, 1946*c*, 1946*d*, 1947; Black, 1945, 1946*a*, 1946*b*; M. J. Mackerras and Ercole, 1947; M. J. Mackerras and Roberts, 1947).

The task of field malaria control and the training and investigation associated with it were part of the responsibilities of the Army Directorate of Hygiene, Pathology and Entomology, which controlled a combined service whose efficiency and enterprise were probably unexcelled in tropical warfare. Though their work was largely unrecognized, our military successes in the Pacific rested directly upon the personnel of the hygiene and associated services. During the earlier phases of the war these were under the divided control of Colonel M. J. Holmes (hygiene), Colonel C. H. Kellaway, F.R.S. (pathology), and Lieutenant-Colonel I. M. Mackerras (entomology), but were later effectively united in a single service. For the greater part of the war this was directed by Colonel E. V. Keogh, to whom the high standard of preventive medicine in the Australian forces was largely due. The valuable work of the army entomologists, which is outlined in recent papers by Mackerras (1947, 1948), has in some part been printed, or is in process of publication.

The intensive investigation of local problems during this period made available, for the first time, comprehensive information upon which adequate practical measures could be based, and upon which peace-time procedures could later be founded. Together with the added advantages of the newer insecticides and drugs, this has greatly enhanced the effectiveness of antimalarial measures in the region.

The Distribution of Malaria in the Australian Region.

In the Australian region the islands of Melanesia, affected to a hyperendemic degree almost in their entirety, form the great reservoir of malarial infection. The disease prevails throughout the mainland of New Guinea and its associated island groups, New Britain, New Ireland, the Admiralty Islands (Manus), the Solomon Islands, and the New Hebrides. In all these islands it forms a major health problem.

To the northward malaria does not exist in the Marshall and Caroline groups. Similarly it does not extend eastward from Melanesia to New Caledonia and the Loyalty group, nor to Fiji, Samoa, Tonga or the other islands of Polynesia. From these islands anopheline mosquitoes have not been recorded. To the west of New Guinea the disease extends to the Indonesian islands of the Oriental Region.

While malaria is not present in the major part of the Australian mainland, foci are found in northern parts of the continent, and conditions of potential danger from its introduction exist over a wide area. The region on the continent so involved, either actually or potentially, corresponds with the distribution of the efficient anopheline vector *Anopheles punctulatus farauti*. In Queensland it extends from the northern coasts southward to 19° south latitude, about the locality of Innisfail; in Northern Territory to about 17° south latitude, between Daly Waters and Newcastle Waters; and in Western Australia to about 19° south latitude, with the exception of a coastal strip up to and including Broome. Throughout this extensive, thinly populated area, owing to the presence of the effective vector species, conditions suitable for the implantation of malaria are likely to arise upon the introduction of malaria carriers.

Over the larger part of the Australian continent south of the potentially malarious area mentioned above, existing conditions are not favourable for the maintenance of the disease. Short-lived and narrowly localized outbreaks have occurred upon rare occasions, though except in a few isolated localities in Queensland, the danger is not significant.

The extent of the malarious and potentially malarious areas within the region is shown in Figure I. These are limited to a zone which lies north of 19° south latitude and west of 170° east longitude.

The Anopheline Vectors.

Prior to the intensive wartime investigations, knowledge of the anophelines of the region was grossly limited, although effective field control had been made possible in Melanesia by the definition of *Anopheles punctulatus* as an efficient vector by Heydon (1923*a*, 1923*b*) and by de Rook (in Swellengrebel and Rodenwaldt, 1932), and by the former author's description of the essential biological characters of the species. Similarly pertinent information was not available for the malarious areas of the mainland until Heydon, in an army investigation of a benign tertian epidemic in Cairns in 1942, proved *Anopheles punctulatus farauti* to be the vector. Subsequent work upon the extent and habits of this subspecies provided a basis for control in north Queensland.

Much of the large body of information collected in recent years yet remains in unpublished reports. Among the anopheline work so far to appear are papers by Bang, Hairston, Maier and Roberts (1947), Lee (1946), I. M. Mackerras (1947), I. M. Mackerras and Aberdeen (1946), M. J. Mackerras and Roberts (1947), Marks (1946), Roberts (1943, 1947), Roberts and O'Sullivan (1948) and Woodhill (1946). A comprehensive monograph on the regional anophelines by Lee and Woodhill (1944) was also published.

In the most important contribution yet to appear on the status of the Australasian anophelines as malarial vectors Mackerras (1947), in the light of the wartime studies, classified the pertinent species in the following order of importance:

Proven dangerous vectors:
Anopheles punctulatus punctulatus Don.
Anopheles punctulatus farauti Lav.

Potential vectors under locally favourable conditions:
Anopheles annulipes Walk.
Anopheles bancrofti bancrofti Giles.
Anopheles subpictus Grassi.
Anopheles amictus hilli W. and L.
Anopheles amictus amictus Edw.

Unknown, but at most locally significant:
Anopheles meraukensis Venh.
Anopheles novaguinensis Venh.

It was held that other species could be safely excluded from consideration.

Anopheles punctulatus is the most dangerous vector in the region. In New Guinea and the Melanesian islands *Anopheles punctulatus punctulatus* and *Anopheles punctulatus farauti* both occur, and are the main vectors. In north Queensland *Anopheles punctulatus farauti* is the chief vector. In addition, localized transmission is considered to be possible under favourable circumstances by certain other species. These, as listed by Mackerras, are shown above.

In the Northern Territory a vector has not yet been determined by dissection. *Anopheles punctulatus farauti*

occurs in the northern parts over a range which is largely coincidental with that of recorded malarial transmission. Although the role of this subspecies is unknown in the area, from its position as a vector in other parts of the region it must be regarded seriously, despite the statement of Woodhill (Mackerras, 1947) that its abundance and behaviour there were never such as to bring it under serious suspicion, whereas certain other species were very abundant and attacked humans with great energy. Some of the latter species are presumably involved in the localized relatively short-lived outbreaks that have been a feature of the disease here, as in Queensland. It is possible, as Mackerras (1947) suggests, that "any of the abundant species will serve, if human populations and parasites are presented to them in sufficient density".

In the southern parts of the continent, *Anopheles annulipes* is the presumptive vector in the sporadic outbreaks and in isolated cases that occur upon rare occasions.

Malaria in Melanesia.

Malaria is prevalent throughout Melanesia, where it is equatorial in type and of perennial transmission, and in many parts is of extreme intensity. The disease forms a serious hazard to the health and development of the native peoples, and is the cause, in wide areas, of a high infant mortality and a lowering of fitness which increases the fatality from other diseases. It exists also as an obstacle to European development, and the conditions pertaining ensure rapid infection of incoming non-immune persons, unless rigorous preventive measures are taken. Fulminating epidemics are probable in collections of non-immunes under such circumstances, of which the devastating outbreaks among Allied and Japanese troops in the Pacific campaigns, and earlier in new settlements and mining camps, are examples.

In the complex of factors that are the cause of depopulation in native communities in parts of Melanesia, chronic malaria plays a large part. For, though other causes have

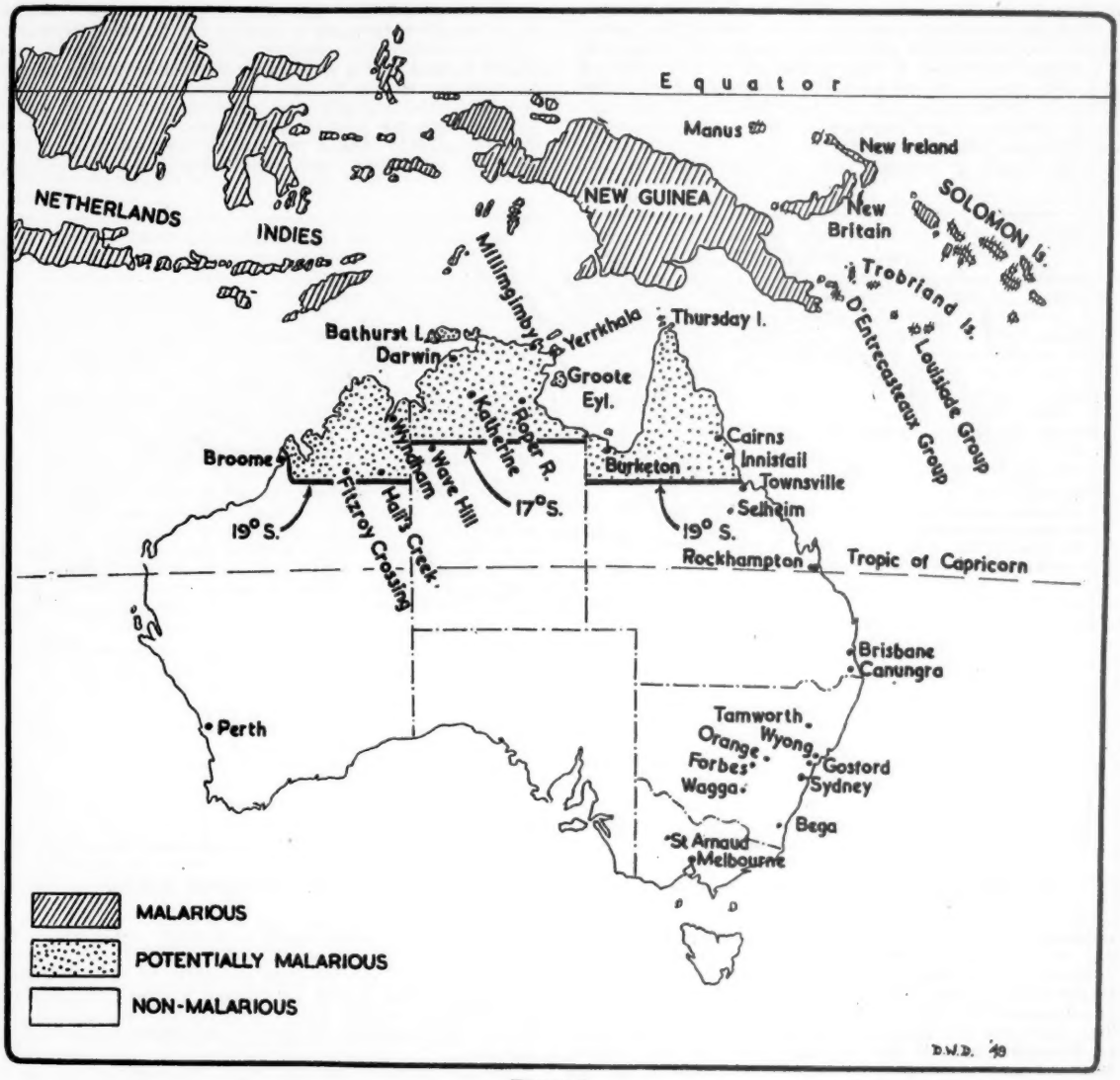

FIGURE I.

Map showing the malarious, potentially malarious and non-malarious parts of the region. Malarial cases of local origin have been reported from places named in the non-malarious area.

been regarded as responsible (Rivers, 1922; Williams, 1933), it appears certain that medical and dietetic factors, rather than psychological or sociological, are the main cause. Cilento (1932) held that diseases, either introduced or accelerated, interacted with food deficiency to constitute a vicious cycle of decline in these islands. In an earlier consideration of the subject in the Western Islands, Mandated Territory of New Guinea (1928), he concluded that, irrespective of secondary factors and sequelæ, the problem could be resolved into those of nutrition and the control of disease, especially malaria. Study of the subject makes it impossible to concur with any other opinion. The psychological and sociological disabilities, frequently claimed as of primary importance in such decline, must be considered as mainly disease effects, as the the states of physical and economic degeneration that are associated with malaria's heavy incidence in any community. The resultant weakness and indigence join in furthering the cycle of decline. Resistance to disease is lowered, only the most essential food

places like Port Moresby, conditions are suitable in the coastlands for transmission to occur throughout the year. A comparable state prevails in the outlying islands under Australian rule: New Britain, New Ireland, the Admiralty Islands (Manus), the great archipelagos scattered to the east of Papua—the Louisiades, the D'Entrecasteaux group and the Trobriands—and also in Bougainville and Buka, the two islands of the Solomon group which are held under the Australian Mandate.

The most dangerous vectors of the Australian Region, *Anopheles punctulatus punctulatus* and *Anopheles punctulatus farauti*, both occur widely in the islands. Owing to their propensity for rapidly extending breeding to artificially formed places such as wheel-tracks, gutters, borrow pits and footmarks, devastating epidemics have occurred in new settlements and alluvial fields, where such waters abound. Similar conditions pertained in the war areas, with comparable results. For though such small collections are easy of control, their multiplicity renders efficient

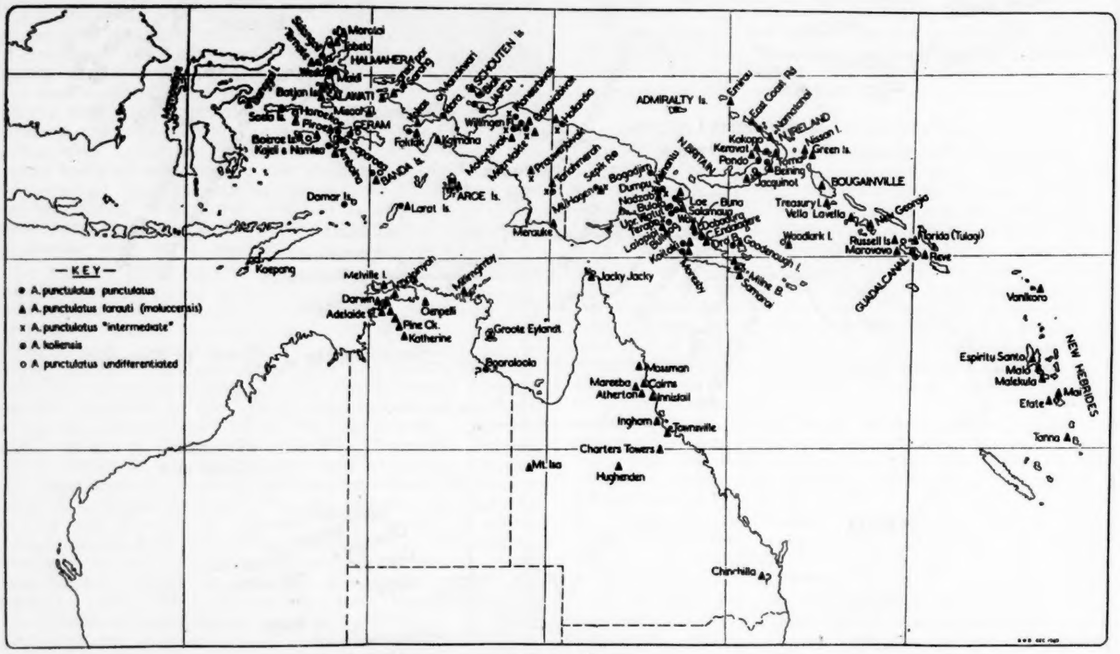

FIGURE II.

Distribution of *Anopheles punctulatus*. (D. J. Lee, School of Public Health and Tropical Medicine.)

production is pursued, and degeneration spreads to every part of the social and economic fabric. The statement by Lambert (1934), that since malaria was always present in the area it could consequently not account for present-day depopulation, disregards the complexity of the subject. The intensity of malaria makes it certain that prior to European contact the physical state of the coastal Melanesians was reduced by heavy chronic infection and its concomitant evils. They were consequently, from the outset of this contact, in a lowered state of resistance to introduced disease. Further, the effect of the transfer of *falciparum* strains consequent on the breaking down of the traditional barriers raised by the old tribal hostilities, and of the possible rise of epidemic states in times of decline, cannot be ignored. The disease is a constantly acting factor, as it is a serious one.

In New Guinea malaria is hyperendemic or highly endemic in the greater part of the coastal region and along the large river systems, as well as on many of the associated smaller islands. The incidence is low in the central highlands and falls to a minimum in the mountainous country above 4000 feet. Except for a few drier

management a heavy task. In both subspecies adult mosquito control is complicated by their resting habits, for though they readily enter dwellings to feed, in general they leave, after biting, for outdoor shelter. Consequently the efficiency both of indoor spraying and of residual DDT is low in comparison with their use against indoor-resting species.

All species of parasites occur, *Plasmodium falciparum* dominating the picture with its high mortality and morbidity in native communities and epidemics among incoming non-immunes, particularly in military operations and new settlements. *Plasmodium vivax* complicates a large proportion of malignant tertian cases, and shows in relapses long after the elimination of *falciparum* infections. *Plasmodium malariæ* occurs sporadically throughout the area, particularly in children. *Plasmodium ovale* has been recorded in only a few cases. Blackwater fever occasionally occurs after repeated *falciparum* infections, most usually in persons suffering from privation and malnutrition.

Among the native peoples, who live under primitive conditions and are for the most part untouched by white

civilization, comprehensive malaria control is beyond existing facilities. But with extended development and the availability of more readily applicable recent methods, organized schemes of partial control may be possible, even with limited resources, in selected areas where most benefit would accrue from some lowering of the malarial intensity.

Great danger exists to unprotected natives from the non-malarious highlands who visit coastal areas, and heavy mortality has at times occurred in these. For this reason such natives, during the war, were prevented by army regulation from being brought into malarious areas for any reason. This policy has also been pursued by the civil administration, and recruiting from the mountain villages for labour on the coasts forbidden. In view of the growing development of the country, and the demands for increased labour, it appears probable that the Administration will not be able to prolong this highly desirable isolation of the highlands for much longer, though the risks can be minimized by carefully supervised protective measures.

White inhabitants are few and exist in scattered settlements and isolated plantations. In 1941 the population of the Mandated Territory of New Guinea included about 5000 Europeans and Asiatics among an estimated 800,000 natives. In Papua there were 1800 whites and an estimated 300,000 natives (Robson, 1942). In 1947 the population, exclusive of the native inhabitants, was in Papua 3239 and in New Guinea 6200, of which 1059 were Asiatics (Census, 1947).

The main towns are now well controlled, but in many outlying places pioneering conditions exist. Formerly, adequate malaria control was found possible by only a relatively few well-established enterprises, but the increased facility and efficiency of recent methods now make this more widely practicable. For effective work constant supervision is necessary, which adds greatly to the expense of all control schemes. A great risk of epidemic subtertian malaria is encountered during early mining and agricultural development, when conditions frequently arise in which there is heavy vector breeding, as well as a close association of infected natives with unprotected Europeans, often in circumstances of privation.

Malaria control in New Guinea was placed on a sound basis by the definition of the vector in 1923. Thereafter, control work in administrative centres was associated with effective surveys with good results (Brennan, 1935). The maintenance of satisfactory control standards rests on the availability of trained personnel for preliminary and routine surveys, and for advisory duties as required. With widely scattered settlement and meagre resources the provision of adequate assistance of this nature is difficult. The recent appointment of trained personnel to the Papua-New Guinea Medical Service, and the extensive wartime experience in the area should, however, ensure a wider availability of these essential facilities.

Malaria in Queensland.

In Queensland, except for isolated cases and rare localized outbreaks, malaria has been limited to the part of the State which lies north of 19° south latitude. In this extensive area the dangerous vector *Anopheles punctulatus farauti* occurs. Ingham and Innisfail are the southernmost centres involved, while Townsville, though close to the affected area, has remained free except for odd cases. Parts of the Atherton Tablelands above 2000 feet, Thursday Island, and Horn Island are unaffected.

The incidence of malaria in these parts has been marked by fluctuations of established *vivax* malaria in certain coastal foci, and by outbreaks of introduced *falciparum* malaria, and at times of *vivax*, that have arisen from time to time in special circumstances.

Benign tertian malaria, usually of low endemicity, persists in scattered parts, almost dying out for periods, and rising at intervals to an epidemic state. Cairns, with a population of about 17,000, is the only large centre so affected, and remains the main focus on the continent.

Subtertian malaria is now absent, though formerly, especially in the pioneering days, it was widespread. The risk of its reestablishment has greatly exercised both public health and army authorities, particularly during the war period. Epidemics have frequently occurred, especially on alluvial mining fields where, as in Melanesia, conditions conducive to epidemic malaria may readily arise. Heavy vector breeding may be set up with rapidity in the artificial breeding waters which abound in such places. Conditions were usually primitive and preventive measures lacking; gametocyte carriers were frequently present from the fact that in every mining rush it was customary to find representatives of the malarious Papuan fields. Consequently many tropical goldfields became death traps. In 1910 such an epidemic occurred at Kidston on the Einasleigh goldfield in north Queensland and was investigated by Dr. Baxter Tyrie of Cairns. This was reported by Elkington (1912), and was typical of many such outbreaks. Subtertian malaria was introduced by two miners from New Guinea and an epidemic condition was rapidly established. In a population of 400 persons, 120 cases, with 24 deaths, occurred before the outbreak subsided. Infection was also common in construction camps during railroad development (Heydon, 1927). Here, heavy anopheline breeding in artificial places similarly occurred under primitive conditions.

Malaria has evidently existed in North Queensland for many years, though from the early accounts, usually by unqualified observers, it is impossible to learn the incidence. Elkington (1912), who perused the death registers of the Gulf country for a period of over thirty years, was impressed by the numbers of deaths which had been returned as due to malaria in the earlier years. He considered that this diagnosis at times covered other conditions, but that the descriptions confirmed the presence of subtertian malaria. The same author described an outbreak in early Burketown which wiped out the greater part of the population of the town, and was the reason for its removal to Sweer's Island, at the mouth of the river. In 1866 a vessel arrived from Java, with some of her crew suffering from what was apparently severe bilious remittent fever. Prior to the arrival of this ship the settlers had been healthy, but within two months some 50 or more persons had died in a population of 76. The disease was believed by residents to be yellow fever, but descriptions left no doubt that it was malaria.

In tropical Australia malaria reached its peak towards the end of last century. At this time, as well as the temporary concentration of population on mining fields, new agricultural country was being opened up in Queensland coastal areas. Cilento and Baldwin (1930) stated that the disease had been reported from almost every area of virgin country under development.

In addition to the coming and going of miners and settlers between Queensland and New Guinea, the native labour trade, established to supply Melanesian workers to the Queensland plantations, was also a prolific source of malarial infection to the State. The traffic commenced about the middle of the last century, and continued till 1904. Native labourers were recruited from the Solomons, the New Hebrides, the Bismarck Archipelago and other malarious islands. Heydon (1927) attributed to the cessation of this system a major part in the lowering of malarial incidence that subsequently ensued. Frequent accounts of the rapid increase in cases upon the introduction of infection to communities in various parts of the Australian tropics, and their waning and apparent total disappearance in the absence of renewed infection give force to this opinion.

As the only urban involvement in Australia, malaria in Cairns has special importance. Though malignant tertian formerly occurred, for some years benign tertian has alone existed, and the recent local history of the disease is mainly concerned with the rise and fall of the latter. O'Brien (1908) reported that though in 1900 malaria was very common in Cairns, by 1908 it had been abolished from the main part of the town by the filling of swamplands. In a district on the outskirts, however, cases still occurred from time to time in almost every house. Ten years later Breinl and Taylor (1918) reported a parasite rate of 13·5% in the examination of 657 white residents, *Plasmodium vivax* and *falciparum* being almost equally

represented. In April and May, 1922, a small epidemic occurred in a restricted area adjoining swampy land, and the next year a survey by the Hookworm Campaign showed parasites in films from 18 out of 554 persons examined in Cairns, and three out of 66 at Yarrabah. Following a later investigation, Heydon (1927) considered that the disease was then absent from Cairns, or nearly so, nor could he find evidence of infection at Yarrabah, Innisfail, Mossman, or the Daintree River, in all of which it had also previously existed.

The last epidemic in Cairns occurred in 1942, when there were over 700 civilian cases of *Plasmodium vivax* infection. As Cairns was at that time the main forward port for supplies to New Guinea, as well as an important staging base for troops, vigorous action was taken by the army. This was concerned with both the control of the civilian reservoir and the protection of personnel from this source, as well as with preventing the introduction of malaria, and especially malignant tertian, to this and other susceptible places. The last-mentioned was not only an important measure as regards the civilian population, but was also of military necessity to prevent wastage in the northern battle stations, which were then of critical importance against threatened invasion.

Since the basic entomological data for effective control were lacking at this time, work was urgently undertaken by Heydon, Roberts and other army workers, and it was shown that *Anopheles punctulatus farauti* was the vector in this area. As a result, intense species sanitation was made possible by army malaria control units, and this was maintained throughout the war period. The work at first consisted entirely of temporary measures, which were later replaced as far as possible by drainage. The 1942 epidemic among civilians waned rapidly, and the incidence thereafter has remained low. In April and May, 1944, in a survey of civilians in the previously epidemic quarter, Captain B. Roberts (unpublished) reported a parasite rate of 0·28% (*Plasmodium vivax* only) and a splenic index of 3·1.

During the war in the Pacific, measures for preventing the introduction of malaria into the susceptible parts of Australia by infected personnel from New Guinea involved a heavy task of organization and administration. This was commenced early in the Papuan campaign, and pursued with complete success throughout the war. Orders were rigidly enforced that, except for reasons of urgent military necessity, units or individuals were not to enter a potentially malarious area within six months of leaving a malarious area. The extent of these areas has been previously described. In Queensland this involved the whole northern part of the State to as far south as 19° south latitude, Cairns being the main centre affected. For the reception, training and hospitalization of infected troops within these limits, non-malarious areas were selected, of which the Atherton Tableland, beyond Cairns, was the most important. Since *Anopheles punctulatus farauti* does not exist there at elevations of over 2000 feet, the danger of introducing the disease was minimized. Infected troops arriving at the port of Cairns for tableland encampments were disembarked in daylight and entrained immediately, so that they moved out of the lowland district prior to the nocturnal biting time of the vector. The troops in the Cairns base itself were selected as being non-infected and were protected from civilian infection by preventive measures. Service personnel who suffered a malarial attack were immediately removed to hospital on the Atherton Tableland. These measures applied to all services of both Australian and American forces, and were for the most part performed carefully. The only exception to these arrangements occurred in places on the coast where it was occasionally necessary to introduce infected troops for special training, such as beach landings, and here the vicinity was controlled. Great inconvenience was involved from the inability to transfer units immediately from New Guinea to areas in North Queensland and the Northern Territory. But even in the most critical period of the New Guinea campaigns it was insisted that the orders be complied with fully, and consequently the plan achieved complete success.

South of Ingham, which lies at the southern extremity of the susceptible area discussed above, malaria occurs only rarely. Localized outbreaks have been reported from various places where conditions for transmission have become temporarily favourable. In 1943 an epidemic of such nature occurred in an army camp at Sellheim, on the Burdekin River. Heavy breeding of *Anopheles annulipes* existed in the vicinity of infected troops from New Guinea, and 28 cases of benign tertian malaria resulted before vigorous control measures by Sinnamon were successful. Under similar conditions, at the Jungle Training School at Canungra, 44 infections of benign tertian malaria were contracted between October, 1943, and April, 1944. Isolated cases also occurred among civilians in various parts, particularly in 1943 and 1944, when large numbers of infected troops were quartered in southern Queensland. In Townsville 11 civilian cases occurred in 1943, and three in 1944; in Rockhampton 35 in 1943 and two in 1944; and in Brisbane 13 and 33 cases for the same years. Smaller numbers were reported from other localities. The incidence fell rapidly as the gametocyte carriers lessened, and little trouble is to be expected henceforth in parts uninhabited by *Anopheles punctulatus farauti*.

In spite of limited antimalarial measures, especially in rural districts, the malarial incidence in Queensland has gradually fallen in recent years. Cilento and Baldwin (1930) ascribed this to the rising economic status of the population, with improvement in the standard of living and hygiene, and increased availability of medical and protective facilities. This has been associated with the progress of agricultural development and with the stabilization of mining. The beneficial effect of the restriction of the importation of native labourers from Melanesia early in the century has already been mentioned.

Further betterment can be expected from the present policy of the Queensland Government in granting a 50% subsidy to all local authorities on approved mosquito eradication and control works. These consist of drainage schemes, reclamation of low-lying land, spraying, and clearing of watercourses. From 1943 to 1948 subsidies were granted to a total of £358,466, of which £142,080 was for the year 1947-1948. From applications received, subsidies for 1948-1949 were estimated to approach £171,000, the total cost of the projected work amounting to double this sum. A large part of the expenditure under this scheme is applied to general anti-mosquito measures and not particularly to anopheline control. Much benefit will be derived, however, especially in the northern communities such as Cairns, where it is considered by Fryberg (1948) that, while the present system of control prevails, malaria epidemics similar to that of 1942 should not be possible. Apart from the large-scale malaria control organizations of the armed forces during the war period, the Queensland Government project is the most significant that has yet been established in the Australian Region.

Technical advice was provided in Queensland by the establishment of a Queensland National Mosquito Control Committee by the State Government in 1943. The Committee consists of the Director-General of Health, the Director-General of Education, and representatives from the University of Queensland and from local authorities. An annual grant is provided by the Government (£850 in 1947-1949) and laboratory and other facilities by the University of Queensland. The Committee undertakes the identification of mosquitoes forwarded by local authorities, and advises on control measures upon request, although the research aspects of the subject form its main consideration. Valuable work on the mosquitoes of Queensland has been carried out by Mr. F. A. Perkins (secretary of the committee) and Miss E. N. Marks. This organization forms an important adjunct to the mosquito control scheme of the Queensland Government.

Malaria in Northern and North-West Australia.

In the Northern Territory and north-west Australia mildly endemic malaria exists in widely scattered districts. Epidemics of subtertian and benign tertian malaria have arisen from time to time, usually on alluvial mining fields, construction camps or cattle stations, where favourable

conditions for transmission may arise upon the introduction of human carriers. Owing to the sparse population of the Territory (0·01 person per square mile), and the great distances between settlements, such outbreaks have usually been localized, though at times they have become widespread.

The disease is most persistent about the larger rivers, including the Roper in the east, the Alligator and others in the north, the Daly and Katherine in the north-west, and the Victoria and Fitzmaurice in the west (Mackerras, 1943; Ford, 1943). These areas characteristically show alternating periods of incidence and of apparent freedom from infection.

Malaria has occurred in various places from the Gulf of Carpentaria in the east to the Ord and Fitzroy River valleys in the farthest north-west, and from the northern coasts south to Katherine and Mataranka. It has at some time occurred in practically every community within these limits. Darwin, the most populous centre, has, however, remained practically free, only two cases, of benign tertian malaria arising locally in the ten years prior to the war. This is due to effective natural drainage, and anophelines are infrequent in residential parts of the town.

An anopheline vector has not been determined by dissection in the Northern Territory. Since no malarial outbreaks occurred there during the war, an opportunity for investigation by army specialists did not arise. *Anopheles punctulatus farauti* has been found at various places on the coast, and extends inland from Darwin as far south as Katherine, its range largely coinciding with the distribution of malaria. This important vector must be regarded seriously wherever it occurs, though it is possible that less efficient anophelines, breeding in large numbers, are often responsible for transmission. As indicated by Mackerras (1947), *Anopheles annulipes* and *Anopheles bancrofti* are the most obvious of these, and *Anopheles amictus amictus, Anopheles amictus hilli* and *Anopheles meraukensis* are also sometimes numerous and of probable significance.

Most of the malarial outbreaks in the Northern Territory have been due to malignant tertian, though benign tertian malaria is relatively not uncommon. All cases contracted locally during the war period were of the latter, and an epidemic at the Roper River Mission in 1945 was similarly benign tertian in form. In earlier times the mortality from *Plasmodium falciparum* malaria was usually heavy on account of the isolation of the sufferers and their distance from medical aid and from ordinary amenities. As in other places where difficult pioneering conditions prevailed, the disease frequently went undiagnosed on isolated fields until survivors reached civilization with accounts of disaster. In 1909 and 1910, when large numbers assembled on newly developed fields, malaria accounted for 25·5% and 20% respectively of the total death rate for the Northern Territory (Breinl, 1912), though frequently the diagnosis was made by police officers and others. The records of this and earlier times often show "fever" as a cause of death, so that the totals of deaths certainly due to malaria are not available.

From consideration of the distribution of the disease, and of the probable vectors, the part of the Northern Territory which lies north of 17° south latitude was regarded by the Australian Army authorities as potentially malarious. This parallel of latitude crosses the north-south road between Daly Waters and Newcastle Waters. In Western Australia the southern limit of the susceptible area was arbitrarily placed at 19° south latitude, except for a strip of coast land extending up to Broome. This division was made for administrative purposes connected with the prevention of the introduction of malaria during the military occupation, which will be mentioned further.

The epidemic experience of the area indicates that widespread conditions occur at intervals during which the introduction of *Plasmodium falciparum* carriers is fraught with considerable danger. According to Kirkland (1939) severe outbreaks are associated with heavily wet seasons, followed by spells of warm, humid weather, together with some abnormal concentration of humans. Transmission usually occurs in the dry season, from May to October, with a maximum incidence usually in May and June, and falling away as the non-permanent water collections are dried up. In the wet season, from October to April, breeding places are flushed by the heavy rains. Under unusual conditions, however, heavy mosquito breeding and consequent transmission have occurred during this season.

Though in general the small communities are separated by great unpeopled distances, which tend to localize infection, malaria may be widely distributed by travellers, drovers, prospectors and aborigines. It has often occurred that upon its introduction to a settlement, the disease has thereafter appeared at intervals in places along the outpassing routes, to which it has been carried, frequently over hundreds of miles, by such agents. Breinl (1912) reported that following a severe subtertian malarial epidemic on the tin-mining field at Umbrawarra Creek in 1909-1910, malaria was in evidence in practically every mining camp and cattle station visited during an extensive survey, both *Plasmodium falciparum* and *Plasmodium vivax* being present. In this outbreak it had been introduced by New Guinea miners who, as in tropical Queensland, were a danger on the alluvial fields, and many epidemics were ascribable to their presence. There was a constant interchange between the goldfields of Melanesia and Northern Australia, and miners were accustomed to transfer, with notorious facility, from one place to another in which fortune appeared more imminent.

An account of the above epidemic by Holmes (1913) probably presents a state that was common on alluvial fields in tropical Australia:

> At Umbrawarra Creek, stream tin was found in good payable dirt. A rush to the field occurred. Hundreds of men came from all parts, and from other countries, and settled . . . right alongside the course of the creek. The camps were formed in close proximity to pools where mosquitoes capable of carrying malaria were breeding in large numbers. Some of the men, coming from malarial countries such as Papua, brought fever with them. Working all day almost to the waist in water under a hot sun, sleeping without nets close alongside the mosquito breeding pools, many of them spending a large portion of the nights in a drunken carouse with all its attending carelessness and exposure, the general health of the men would be quickly lowered. . . . Nothing more favourable to the spread of malaria could be imagined than the conditions in a large mining camp situated alongside a shallow creek, which is stagnant for the greater part of the year, thus affording every opportunity for the rapid multiplication of mosquitoes.

The most recent epidemic, which occurred from 1928 to 1934, and in which subtertian malaria jumped from place to place in a severe progress from the Gulf of Carpentaria westward to the north-west coast, indicates the wide distribution possible under favourable circumstances, despite the small and scattered population. In 1930 the white population of the Northern Territory was 4616 and the estimated number of aborigines and half-castes 21,800. A large proportion of these were situated outside the malarially affected area, as at Darwin and in southern parts.

In 1928 subtertian malaria occurred in the Roper Valley, near the Gulf, and extended to Tanumbarini and Nutwood Downs. In 1929 Cook found a high incidence in the area. Vigorous action was taken by the Northern Territory Medical Service, but in 1930 cases occurred in widely separated stations to the westward, and at places between Darwin and Katherine. By this time the Roper Valley was practically free of cases.

In 1931 and 1932, despite the efforts of the medical services, hundreds of cases occurred in scattered places to which infection had extended, including an outbreak in 1931 in the Katherine-Mataranka district, where 279 cases were reported with a comparatively high mortality. This followed an unusually heavy wet season, after which exceptionally good conditions for transmission prevailed. The time was one of industrial depression, when unemployment and poor living conditions were common. In 1933 outbreaks occurred in the Wave Hill and Victoria River districts, still further to the west (Cook, 1929, 1930, 1931, 1932, 1933, 1934; Kirkland, 1939).

In 1934 epidemic conditions extended to the Ord and Fitzroy River valleys in the north-west. The severity of the disease in this area is shown by the fact that nearly 200 aborigines and 15 whites died in the Fitzroy Crossing district, and it is certain from unreported native deaths that the total was much higher. In the early stages of the outbreak here the disease was regarded as influenza. Subtertian malaria had not been previously reported in Western Australia, though benign tertian malaria was known in these parts. The epidemic rose here in the wet season, when the district was largely cut off from communication by widespread flooding. Consequently great difficulty was experienced in the application of preventive measures, though a medical officer was able to be landed by plane (Atkinson, 1935). In the following year the incidence dwindled over the vast areas affected and only a few cases were reported. Parasite rates of 2%, 4·4% and 1·8% were found at Victoria River Downs, Wave Hill and Ord River respectively (Kirkland, 1935).

It seemed reasonable to assume that the collapse of the epidemic was mainly due to the intensive measures undertaken and more especially to the "Plasmoquine" treatment of carriers found on the repeated comprehensive surveys of the limited population. In support of this Kirkland (1939) noted that in the Daly River district, where such measures were not carried out, cases continued to occur after adjoining treated areas were free. But since this epidemic followed the pattern of numerous less extensive incidents, where a similarly rapid apparent disappearance of infection had occurred in the absence of such well organized measures, it is probable that the campaign was aided by a return to less favourable conditions for transmission.

Various opinions exist as to the importance of the uncivilized aborigines as a reservoir of infection, and valueless conclusions have frequently been drawn from single examinations of small groups. It is improbable that the disease is firmly established in any of the bush tribes, and likely that in general they are subject to the fluctuations of incidence that occur among other inhabitants. Though they are few in numbers and are in infrequent communication with settlements, they were held by Kirkland (1939) to be important agents in malarial dissemination. For apart from their wandering existence being conducive to this, natives in employment are probably infected from them during walkabouts and convey the parasites to their place of work. Despite a common opinion to the contrary, as expressed by Basedow (1932), in general there is no evidence of immunity to malaria in the natives of the northern coasts.

Coastal natives are liable to contract malaria from the crews of pearling vessels. Formerly a risk existed from the Malay and Macassar fleets that annually crossed the Arafura Sea on fishing and trading expeditions. The traffic ceased in 1904 upon the introduction of Commonwealth legislation. It was insisted that such vessels, which had previously been inspected at the Bowen Strait by South Australian officials, should proceed to Darwin for examination. This was rendered difficult for the sailing craft by the prevailing monsoon, and the long-established trade fell away (Conigrave, 1936).

Malaria at times occurs in outlying mission stations, which are frequently visited by natives, who make them points of call on their long journeyings. Consequently, infection in either station or bush natives is likely to reach the missions eventually, with a risk of local transmission and further distribution in favourable circumstances. A persistent focus exists about the Roper River Mission, where cases have occurred for many years. Breinl (1912) reported the presence of benign tertian malaria, and the widespread subtertian epidemic of 1928-1935 commenced in this area. In 1939 malaria was apparently absent, and an examination by C. C. Fenton and Ford of 58 aboriginal children and 59 adults showed no splenic enlargement, and no malarial parasites in 25 thick blood films. An army survey of this area in 1945, however, showed a splenic index of 25 and a parasite rate of 33% (*Plasmodium vivax*).

Though malaria had been reported earlier from Oenpelli, on the East Alligator River, Ford and Fenton found no signs of its presence at the Mission in 1939. An examination of 29 adult natives and 23 children revealed no splenomegaly, and parasites were not found in films from the latter. An army survey there in 1943, reported by Captain A. N. Woodhill (unpublished), showed no splenic enlargement in 35 native children, and moderate splenomegaly in two adults of 40 examined. No parasites were found in thick films from the latter. It was reported that in ten years no member of the Mission staff had suffered from malaria. The district has not, apparently, the importance as an endemic focus ascribed to it by Ford (1943).

At Millingimbi, in the Crocodile Islands, off the northern Arnhem Land coast, Fenton and Ford in 1939 similarly found no signs of malaria in 63 natives in the vicinity of the Mission. In an army survey there in 1943, thick films from 43 adult aborigines and 34 children were "negative", and splenomegaly was present in only one adult of this group (Woodhill, unpublished). In 1939 the disease was also inactive in the vicinity of the mission stations at Yirrkhala, in the extreme north-east of Arnhem Land, and at Groote Eylandt, in the Gulf. In the former Ford and Fenton found no traces of malaria in 69 natives of various ages, some of whom had recently arrived from the Caledon Bay and Blue Mud Bay districts. On Groote Eylandt, among 48 adults and 59 children examined, one child was found with splenic enlargement of lesser degree. No malarial parasites were found in thick films from the later or from 20 other children.

Though frequently reported from Bathurst and Melville Islands, malaria is at present apparently absent. The personnel of the first white settlement in Northern Australia, which was established in 1824 at Fort Dundas, on Melville Island, suffered severely from what appears to have been malaria during the five years that elapsed before the site was abandoned. In 1911 a subtertian epidemic on Melville Island caused some 30 deaths in natives (Kirkland, 1939), and a further subtertian outbreak in 1912 was reported by Holmes (1913). From a survey Breinl and Holmes (1915) concluded that malaria was then absent, and a later survey in 1939 by Ford (1942) showed no indication of infection. It would appear with these islands, as with many parts of the mainland, that malaria when introduced tends to disappear in a relatively short time in the absence of reintroduction of the parasite, and a regular importation is necessary for its maintenance. With the cessation of the annual visits of Macassar crews the islands were largely isolated from such renewed infection. Although there is at present a constant traffic of the islanders to and from the mainland, this is almost wholly with Darwin, which is free of malaria.

The army malaria control policy during the military occupation of Northern Australia relied mainly upon the exclusion of gametocyte carriers from the potentially malarious area whose limits are described above. Since most Australian front-line troops were engaged in malarious areas, and yet the strength of the Northern Territory force remained of strategic importance on account of the threat of invasion, this scheme offered great difficulties to commanders during the changing fortunes of the period, when free troop movement was desirable. The plan was, however, rigorously maintained from its inception. Units and individuals were not allowed to enter the prescribed area for six months after leaving a malarious place. In the case of key personnel for whom this restriction had at times to be relaxed, suppressive treatment was taken for the whole period of service in the area, weekly blood examinations were made, and special protective measures were required. Malarial patients were immediately removed by air from the area, and rigid measures were taken in the locality from which they originated. The vigorous quarantine system was associated with vector control in limited areas and with careful siting of all installations. In addition aborigines were removed from all occupied localities. The efficacy of this scheme is indicated by the almost complete absence of malaria among troops for the duration of the occupation.

Except for the work of the armed services, vector control has been pursued to only a minor extent in north Australia.

Epidemics in outback places have been dealt with in recent years by the Northern Territory Medical Service with good effect, chiefly by instruction on camp siting and protective measures throughout the area, by segregation of aborigines and by the control and treatment of malarial carriers. Aerial medical services were widely used. Effective control was also maintained during railway construction, when large gangs were concentrated in areas in which serious epidemics had previously occurred. An experienced medical officer was appointed for this task, and success was considered to be due to careful camp siting, to the prohibition of aborigines from the construction area, and to the good living conditions provided, although seasonal conditions favoured the work (Kirkland, 1939). At present, though the risk to life has been lessened by the wider distribution of medical care and by radio and flying doctor facilities, the danger remains of a wide spreading of malaria under suitable circumstances. Prompt action and adequate resources are then necessary to cope with the situation.

Malaria in Southern Australia.

Over the larger part of the continent, to the south of the potentially malarious tropical areas, malaria has rarely occurred and conditions are unfavourable for its establishment. This non-malarious zone, which contains all the larger centres of population, lies to the south of 19° south latitude. After the two world wars there occurred a large influx of human malaria carriers over this area, in places associated with heavy anopheline populations, especially of *Anopheles annulipes*. The extremely low malarial incidence resulting from this association provided evidence that, except in certain limited areas in Queensland in which favourable conditions may at times arise, even the introduction of large numbers of gametocyte carriers entails no risk.

The possibility of the establishment of malaria in southern Australia concerned Cumpston and other health authorities for many years. Cleland (1915) considered that a grave danger existed in certain areas where *Anopheles annulipes* occurred. In 1917 the return of infected soldiers led to an investigation by the Commonwealth Health Department into this risk, particularly in the irrigation areas about the River Murray and its tributaries (Taylor, 1917). *Anopheles annulipes* was found to be prolific in these parts and the situation was viewed with concern. In 1919 projected soldier settlement in irrigation areas again alarmed experienced writers (Evans, 1919; Fowler, 1919). The Federal Quarantine Department was given the responsibility of controlling and coordinating measures for preventing the spread of the disease by returned servicemen, and a scheme was set up to this end (Cumpston, 1920). The cooperation of the State Health Departments was sought in regard to the notification of cases and the adoption of a general system of control.

By 1921, from the few cases arising under these circumstances, the view was expressed by Evans (1921) and Ferguson (1921) that there was little danger that malaria would become generalized in these areas. This opinion has been confirmed by later experience, especially that associated with the more recent war, when even more stringent tests were imposed and wider areas subjected to similar trial.

The implication of *Anopheles punctulatus farauti* as the vector in North Queensland in 1942, the definition of the southern limit of its range, and the subsequent tentative opinion that this species could be regarded as the only constantly dangerous vector on the Australian mainland, enabled a boundary to be set with some assurance between the southern division of the continent in which the implantation of malaria is unlikely, and the northern division where conditions favourable to its existence commonly occur.

As mentioned, in isolated cases malaria has been locally contracted in southern parts of the continent, though these cases have been infrequent, considering the chances for transmission that have abounded, especially where *Anopheles annulipes* occurs in large numbers. In New South Wales single indigenous cases have been reported from Gosford (Jamieson, 1915), Wyong (Evans, 1919) Wagga district, Tamworth district and Forbes (Clayton and Utz, 1921), Bega (Tebbutt, 1942), Orange (Matthews, 1946), and Sydney (Money, 1926; Cunningham, 1946). In Western Australia a single case of this kind was reported from Maylands, near Perth (Baldwin and Cooling, 1920), and in Victoria cases from Saint Arnaud (Doyle, 1921) and Melbourne (Mackenzie, 1948). Further cases also occurred during the war years, when large numbers of gametocyte carriers were widely introduced. *Anopheles annulipes* is the presumptive vector in these southern parts. No malarial infections of local origin are known to have arisen in South Australia or Tasmania.

Basic Problems on the Australian Mainland.

Introduction of Malaria to Susceptible Areas.

On the Australian mainland the northern region, rendered potentially malarious by the occurrence of the efficient vector *Anopheles punctulatus farauti* and by conditions frequently favourable for transmission, lies in proximity to the hyperendemic Melanesian reservoir. The introduction of malaria carriers is frequent and in ordinary times is not controllable. For though in wartime service organization was successful in this, it relied on powers which are fortunately rarely available.

Widespread implantation of the disease has been counteracted by sparseness of population, apart from other probable factors, though it is endemic in certain districts, and serious outbreaks have occurred in aggregation under primitive conditions of settlement. Under recently pertaining conditions, however, malaria has receded from some districts and with methods now available can be more readily combated. Nevertheless a serious problem awaits further settlement of the north.

Control of Existing Endemic Foci.

The existing foci of malarial infection in tropical Australia, as well as showing a smouldering incidence and occasional serious outbreaks, are reservoirs from which spread threatens susceptible areas.

Control is usually difficult on account of the scattered population involved and the usually insufficient resources for organized work. But the important Queensland Government scheme for granting 50% subsidies to local authorities on approved antimosquito projects, and the effective work of the Northern Territory Medical Service against outback epidemics in recent years, are substantial foundations for future measures.

At Cairns, the main endemic centre, malaria was practically extinguished during the war years by concentrated army work, and this satisfactory condition has since been maintained by subsidized local efforts. Malaria is at present restricted to benign tertian in this district; the possible reintroduction of malignant tertian malaria raises a more serious problem.

Establishment of New Vectors.

The possible introduction of new anopheline vectors from outside Australia is a great danger to the northern tropical zone. The problem, which remains a constant concern to health authorities, can be adequately met only by careful routine work—spraying of aircraft in passage and upon arrival, aerodrome control and periodic surveys of the areas of entry. Dangerous anophelines have been frequently collected by Commonwealth Health Department officers from incoming aircraft at Darwin. These have included *Anopheles sundiacus*, considered by Christophers (1933) to be one of the most effective vectors known, *Anopheles maculatus*, another important vector in Malaya and the Netherlands East Indies, *Anopheles aitkeni* and *Anopheles vagus*.

The possible establishment of *Anopheles sundiacus*, from the Netherlands East Indies, is a serious hazard. Apparently suitable breeding places for it abound along the northern coasts. Its brackish water breeding is difficult of control, and its implantation would raise a problem whose magnitude would become fully apparent only as the Territory developed.

Another special danger exists in the introduction of *Anopheles punctulatus punctulatus* from New Guinea. In Melanesia this vector, though occupying low country similar to that generally preferred by *Anopheles punctulatus farauti*, with which it often coexists, also extends to higher altitudes. There is consequently a danger, in the event of its introduction into the mainland, that it may occupy not only the lowlands at present rendered potentially malarious by the presence of its related subspecies, but also the tablelands near the coast in which efficient vectors do not at present exist. There would also be a risk of its spread southward into areas in which there are at present no effective vectors.

The possibility of the extension of *Anopheles punctulatus* from its Melanesian habitat to the non-malarious islands of Fiji, Samoa or New Caledonia, or northwards to the uninfected Micronesian groups, is also a subject of great importance (Lambert, 1928; Hermant and Cilento, 1920). Though Taylor (1943) held that there was slight chance of this, it is considered that these islands are threatened by invasion with this or other vectors, and that the prevention of such extension is one of the most important public health tasks in the Pacific.

The Risk in Non-Tropical Australia.

Though the possible establishment of malaria in the more temperate parts of Australia has been regarded with concern, little danger of this exists south of 19° south latitude, where *Anopheles punctulatus farauti* does not occur. Exception must be made in the case of restricted areas in Queensland, where self-limited outbreaks may be caused, under exceptional circumstances, by less effective vectors.

BASIC PROBLEMS IN MELANESIA.

Reduction of Malarial Intensity in Hyperendemic Areas.

In Melanesia malaria in the native population has been regarded as largely beyond control. Its widespread and heavy incidence, the meagre economic facilities, and the primitive standard of civilization pertaining provide a task which might well appear to be beyond practical solution, though amelioration is now possible by recent methods.

For a pertinent effect from limited resources, antimalarial measures are required in selected places where the inroads of the disease are most serious. In such places complete control is not necessary for worthwhile alleviation, and any method which lowers the malarial intensity to a significant degree must be of value. Expensive measures are not feasible, and schemes requiring constant and unsupervised attention by natives themselves have little chance of success.

The only method of extensive village control attempted before the war was the introduction of *Gambusia* to anopheline breeding places. In 1930 hatcheries were established at Rabaul by the New Guinea Medical Service and fish were widely distributed. This was stated to be the most successful antimalarial measure in the Territory to that time (Brennan, 1935). Satisfactory results were also obtained by Holland (1933) over a New Ireland coastal area 75 miles long.

Of the newer methods, DDT spraying for residual effect appears to offer most advantage. Field experiments in Papuan villages gave promising results (Bang, Hairston, Maier and Roberts, 1947), though the striking effects obtained in other regions against indoor-resting anophelines are not to be expected on account of the habits of *Anopheles punctulatus*, which usually allow only minimal contact with treated surfaces.

The use of "Paludrine" and other recent drugs offers further means of control, especially of the severe epidemics that at times occur. Field investigations are required to find whether more than occasional use should be made of these among immune natives in circumstances where supplies and distribution are assured (Fairley, 1946).

Apart from economic facilities, the success of such measures depends on an increasing emphasis on preventive medicine by tropical services.

Protection of Non-Immune Natives.

The danger to non-immune natives from New Guinea mountain districts in visiting the malarious coastlands, and the adequate safeguarding of these at present exercised by the Administration through legislation restricting their recruitment, have been mentioned. When this isolation of the highland peoples can no longer be maintained, a protective scheme for recruited labourers can be readily instituted.

Control in White Settlements.

The difficulties encountered in achieving control in the scattered settlements have already been considered. As in the case among natives, special provision for preventive work is necessary, and trained antimalarial personnel under an expert malariologist are required. The recent appointment of such officers to the Papua and New Guinea services must prove of great value.

SUMMARY.

1. An account is given of the distribution of malaria in Australia and its Pacific dependencies, with some details of the extent of the problem in the various parts of the region.

2. On the Australian mainland problems arise from the nearness of hyperendemic reservoirs of infection to a vast northern zone which is rendered susceptible by the presence of the efficient vector *Anopheles punctulatus farauti*. Parts of this area, which extends south to 19° south latitude, are subject to low endemicity and occasional epidemics.

3. On the mainland south of this potentially malarious zone, except for limited areas in Queensland, implantation of the disease is improbable.

4. A serious danger exists of the introduction of new anopheline vectors to northern Australia.

5. In Melanesia the basic malarial problems may be stated: (*a*) the reduction of the malarial intensity in hyperendemic areas to an extent which will lower the infant and child mortality and lessen the effects of chronic infection among the native inhabitants; (*b*) the protection of non-immune natives from infection and consequent mortality, through exposure in malarious districts; (*c*) the protection of European settlers and the prevention in new settlements and industrial ventures of the subtertian epidemics for which Melanesia is notorious.

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TUBERCULOSIS MORTALITY OF CHILDHOOD IN AUSTRALIA.

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MORTALITY from tuberculosis (all forms) in Australia has already been discussed (Lancaster, 1950). In the present paper the discussion will be confined to the disease in childhood, and some analysis of the deaths by form (that is, system) and by type of organism will be attempted.

Sources of Data.

The sources of data, chiefly "Demography", have been described in the previously mentioned article. In order to obtain mortality rates, we need in addition to the population estimates in the usual five-year age groups, published or prepared by the Bureau of Census and Statistics, Canberra, the populations by single years of age for the first five years of life. For any period we have computed the tuberculosis mortality rate of children under one year of age by dividing the deaths from tuberculosis of children under one year of age by the number of births in the corresponding period. This is in conformity with the usual practice of computing the infantile mortality rates, of which these tuberculosis deaths form a part. For the ages one, two, three and four years last birthday, divisors have been computed from the recorded statistics of the births and of the deaths of children for the first five years of life by means of an accepted method, migration being neglected. Our estimates are, therefore, probably accurate for the periods 1911 to 1920, 1931 to 1940 and 1941 to 1945 when the net migration was small, but less accurate for the periods 1908 to 1910 and 1921 to 1930, when the rate of net migration was higher.

In any age group, however, it is unlikely that the difference of our estimates from the true value would exceed 2%.

Forms of the Disease.

I shall consider tuberculosis by forms or systems, so I shall first briefly mention the meanings of the terms used.

In accordance with the coding manual of the Registrar-General for England and Wales (1931), "respiratory" tuberculosis will include all deaths from tuberculosis of the tonsil, pharynx, nose, larynx, lung and hilar lymph nodes, but will not include deaths from tuberculosis of the cervical lymph nodes. The term "nervous" will be used to mean "of the central nervous system". "Alimentary tuberculosis" includes tuberculosis of the alimentary tract beyond the pharynx and the related lymph nodes. Deaths from tuberculosis of the cervical lymph nodes, however, are included in those from tuberculosis of the lymphatic system, which has been shown as part of other "systems". Deaths from all "generalized" tuberculosis have been consolidated under one heading, as there seems little justification for treating them separately as "acute", "chronic" or "not stated", at any rate in children.

Deaths from Tuberculosis (All Forms).

In Table I are set out the deaths from "tuberculosis (all forms)" by single years of life for the first five individual years of life. These are then totalled to give the deaths in the first five years of life and the age group 0 to four years last birthday, and the deaths for each of the next two five-yearly age groups are also given in order to complete the deaths from tuberculosis in childhood.

The Age-Specific Death Rates.

In Table II are given the mortality rates from tuberculosis (all forms) as rates *per annum* per million of children at risk. In a single calendar year, the number of children at risk has been taken to be the mean number of children of the appropriate age and sex, alive in that year. The numbers at risk for a given period of years have been computed by summation from the estimates of the appropriate population for each calendar year in the

TABLE I.

Deaths from Tuberculosis (All Forms) for Each of Five Periods.

Age Groups. (Years.)	Males.					Females.				
	1908–1910	1911–1920	1921–1930	1931–1940	1941–1945	1908–1910	1911–1920	1921–1930	1931–1940	1941–1945
0	170	356	219	115	65	124	266	187	84	39
1	119	335	232	146	65	104	261	211	119	59
2	56	210	147	86	40	44	185	124	73	20
3	35	109	96	45	19	27	127	75	46	24
4	33	91	74	49	17	18	89	67	43	8
0 to 4 ..	413	1101	768	441	206	317	928	664	365	150
5 to 9 ..	98	291	216	129	47	87	285	196	130	59
10 to 14 ..	101	252	194	128	39	129	352	254	153	58

TABLE II.

The Age-Specific and Sex-Specific Mortality Rates from Tuberculosis (All Forms).[1]

Age Groups. (Years.)	Males.					Females.				
	1908–1910	1911–1920	1921–1930	1931–1940	1941–1945	1908–1910	1911–1920	1921–1930	1931–1940	1941–1945
0	967	530	319	192	(173)	756	416	287	(148)	(109)
1	769	553	363	256	(194)	710	446	342	217	(183)
2	(374)	354	231	(150)	(125)	(311)	322	204	(133)	(65)
3	(240)	188	(154)	(78)	(62)	(197)	226	(124)	(83)	(81)
4	(234)	(160)	(119)	(84)	(57)	(130)	(162)	(112)	(77)	(28)
0 to 4 ..	553	387	244	155	126	418	332	217	133	95
5 to 9 ..	(151)	111	70	43	(34)	(134)	109	65	44	(44)
10 to 14 ..	165	106	65	40	(27)	211	148	87	50	(42)

[1] The mortality is expressed as a rate *per annum* per million.
The mortality rates by single years for the first years of life have been computed from estimates derived from the published statistics of births and deaths at ages 0 to four years. The mortality rates for the three five-year age groups have been computed from the population estimates supplied by the Bureau of Census and Statistics, Canberra.
The parentheses indicate that the rates are founded on less than 100 deaths.

TABLE III.

Tuberculosis as an Important Cause of Mortality in Childhood. (The Percentage of Deaths from All Causes, that is Due to Tuberculosis.)[1]

Age Groups. (Years.)	Males.					Females.				
	1908–1910	1911–1920	1921–1930	1931–1940	1941–1945	1908–1910	1911–1920	1921–1930	1931–1940	1941–1945
0	1·18	0·71	0·52	0·43	0·45	1·11	0·69	0·52	0·42	0·35
1	5·00	3·65	3·23	3·70	3·56	5·11	3·33	3·55	3·53	3·93
2	6·23	5·51	4·89	4·13	4·24	5·31	5·57	4·95	4·18	2·82
3	6·18	4·43	4·58	2·91	3·02	5·29	5·79	4·34	3·80	4·47
4	7·82	5·04	4·72	4·07	3·07	4·64	5·05	5·07	4·13	1·87
0 to 4 ..	2·21	1·63	1·38	1·24	1·11	2·12	1·74	1·54	1·33	1·06
5 to 9 ..	7·03	4·96	4·06	2·80	2·54	7·01	5·49	4·60	3·85	4·58
10 to 14 ..	9·03	6·28	4·66	3·29	2·41	13·87	10·55	8·18	5·91	5·61

[1] The tuberculosis death have been divided, for each age group, by the corresponding number of deaths from all causes, and the resulting ratio has been mutiplied by 100 to obtain a percentage.

period. The divisors for the first year of life, as mentioned above, are the births over the same period and not the years lived at the age 0 year last birthday. The sums of the populations by single years of life are not strictly equal to the population of the first five years of life as a whole, since this has been computed by the Bureau of Census and Statistics, Canberra, by means of a different method of estimation; but the differences are not great. However, it will be noted that the rate for the age group 0 to four years last birthday is as a rule approximately equal to the average of the rates for the first five years of life considered individually. A great fall in the death rates from tuberculosis is to be noted over the period studied here—namely, 1908 to 1945. This fall is also to be noted in Figures IA and IB, which have a "semi-logarithmic" scale, so that the vertical scale of the death-rates is logarithmic and the horizontal time scale arithmetic. Therefore, the parallel decline in the graphs of the rates for each sex in each of the three five-year age groups shows that the relative rate of decrease is the same in all these age groups in the two sexes. This comparison of the death rates of the same age group over a period of time is the second of the methods which was discussed in our previous article (Lancaster, 1950).

TABLE IV.

The Number of Isolations of the Two Types of Bacillus, Human and Bovine.
(From Table I of Penfold, 1924.)

Site of Disease.	Isolations.		
	Human.	Bovine.	Total.
Cervical glands	2	1	3
Brenchial glands	9	2	11
Mesenteric and portal glands	4	6	10
Other glands	2	0	2
Lung	10	2	12
Cerebro-spinal fluid ..	4	1	5
Other sites	17	0	17
Total	48	12	60

The Mortality for a Given Calendar Period by Age.

In Figure II are compared the mortality rates from tuberculosis by age in childhood in the two sexes for the period 1911 to 1920. The maximum rate of mortality in childhood in both sexes occurs at approximately the end of the first year of life. In both sexes the mortality rates then decline rapidly to reach a minimum in the years about the eleventh year of life. In the males a minimum is reached

TABLE V.

The Type of Tubercle Bacillus in Cases of Cervical Adenitis. A Summary of the Findings of R. Webster (1932).

Type of Bacillus.	Age. (Years Last Birthday.)								Total.
	0	1	2	3	4	0 to 4	5	10	
Human	0	1	2	0	2	5	4	0	9
Bovine	1	2	3	1	1	8	6	3	17
Total	1	3	5	1	3	13	10	3	26

in the age group ten to fourteen years, in the females in the age group five to nine years. This adolescent rise in the female death rates has already been commented on in the general paper, and is a feature of the female tuberculosis rates in many countries. This method of comparison of the mortality by age for a given calendar period is the first method of our previous paper and is equivalent to reading Table II by columns. Since we are dealing with only a restricted part of the life cycle, this method is not likely to lead to fallacious conclusions, such as have often been made when the death rates of the whole life span are considered.

The Comparison of the Male and Female Rates at a Given Epoch.

If we consider the rates at individual ages for the two sexes for a given epoch or period, we find from Table II that the male rates are generally higher than the female rates in the first five years of life. In the age group five to nine years the two rates tend to equality. In the age group ten to fourteen years, the adolescent rise is apparent in females, and so the female rate is higher than the male. A more detailed discussion of this has already been given in the more general paper on tuberculosis mortality. An analysis of the deaths by organs shows that this adolescent rise in the rates for females is due to respiratory forms of the disease. (Compare mortality rates in Tables XI and XII.)

Tuberculosis as an Important Cause of Mortality in Childhood.

From Table III of the previous article it is apparent that tuberculosis is an important cause of mortality in the age groups above the age of five years, but for ages under five years its importance is obscured by the fact that the divisor, total deaths, contains so many infantile deaths. We may therefore express the deaths from tuberculosis as a percentage of deaths from all causes in the single years of life, as in Table III, and note that, above the age of one year, tuberculosis must be among the more important sources of mortality, since it accounts for about 5% to 6% of the total mortality at every year at ages one to four years last birthday. For example, in the period 1911 to 1920 for males, we find that when all deaths under five years of age are pooled, then tuberculosis accounts for some 1·63% of the mortality. But when the deaths are analysed into single years of life, the tuberculosis deaths are seen to be only some 0·71% in the first year of life but some 5% at the ages one to four years. Tracing through the percentage of deaths due to tuberculosis at a fixed age for the various periods, we find that in the later periods the proportion of the total deaths due to tuberculosis is smaller than in the earlier periods—that is, the mortality from tuberculosis at these ages is falling more rapidly than the mortality due to all other causes. Comparisons in Table III may be made by rows, but false conclusions may easily be drawn by making comparisons by columns.

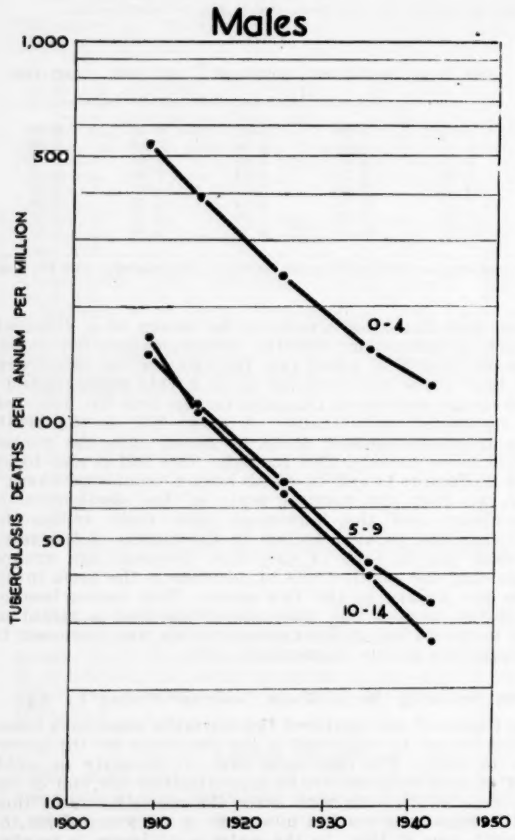

FIGURE IA.

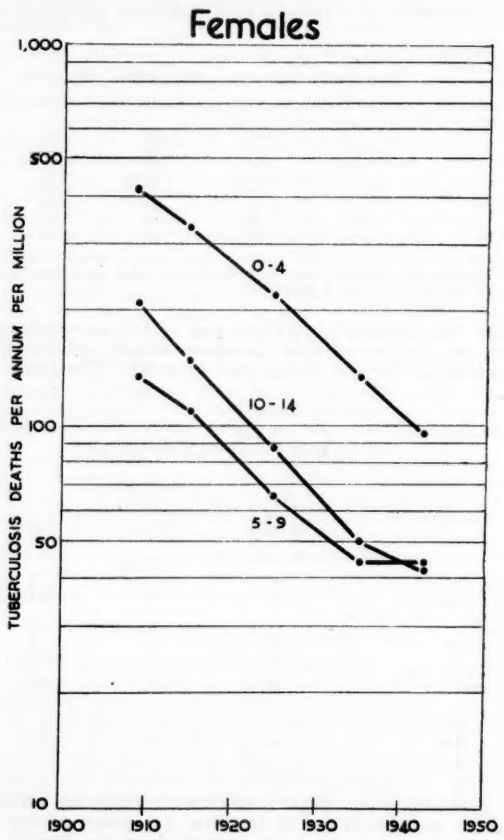

FIGURE IB.

The death rates from tuberculosis (all forms) for children in three age groups for each of the sexes. Semilogarithmic grid.

Types of the Tubercle Bacillus.

Of course, the mortality statistics can tell us nothing directly of the incidence of the two types of bacillus, human and bovine. After a dissection of the deaths by system, however, it is apparent that some indirect inferences may be made, for it is to be noted that tuberculosis of organs in which the incidence of bovine tuberculosis may be presumed to have been high, has fallen more rapidly than that of organs predominantly affected by the human type. W. J. Penfold (1924) and R. Webster (1932) have both published the results of surveys into the type of tubercle bacillus in the disease occurring in childhood in Melbourne. In Table IV Penfold's results are briefly summarized from his Table I. In tuberculosis of the mesenteric and portal glands only was the bovine type the more frequent type of tubercle bacillus isolated. In Table V, however, we see that Webster (1932) has found the bovine type to be the more common in tuberculosis of the cervical lymph nodes. (The material is possibly not strictly comparable, since Webster and his colleagues were making a special search for the bacillus in the cervical glands with the aid of biopsy.) In Table VI, from data in Webster's paper, we find that in the tonsil, too, the bovine type was in a slight majority (four out of seven). Table VII indicates that in Webster's series the proportion of bovine bacilli isolated was approximately the same in each age group. Although each of the two series represents obviously a great amount of work, when the cases are dissected by age and by organ the numbers become small, and so it is not advisable to draw unduly dogmatic conclusions from them. We may assume, provisionally, with the results of other surveys in mind (in England and in the United States of America), that the alimentary forms may be predominantly due to the bovine type, the respiratory forms due to the human type and the meningeal and other forms of tuberculosis of the central nervous system due to either the human or the bovine type of bacillus in uncertain proportion. We are now able to interpret Table VIII, in which the mortality from tuberculosis under the age of five years is shown analysed into the mortality due to tuberculosis of individual systems.

TABLE VI.
(From Webster (1932), Table III.)

Source of Material.	Number of Isolations.		
	Human Type.	Bovine Type.	Total.
Biopsy :			
Tonsils	3	4	7
Cervical glands	7	17	24
Mesenteric glands	0	1	1
Bones	12	0	12
Joints	10	0	10
Cerebro-spinal fluid	2	0	2
Autopsy :			
Bronchial glands	21	0	21
Mesenteric glands	0	3	3
Lung	6	0	6
Total	61	25	86

TABLE VII.
Isolations of Tubercle Bacilli by Age of Patient.
(From Webster (1932), Table V.)

Age. (Years.)	Number of Isolations of Tubercle Bacilli.		
	Human Type.	Bovine Type.	Total.
0	37	12	49
5	17	6	23
10 to 14	6	3	9
Total	60	21	81

TABLE IX.
The Deaths from Tuberculosis at Ages Five to Nine Years Analysed by Form.

Form of Tuberculosis.	Males : Period			Females : Period		
	1911–1920	1921–1930	1931–1940	1911–1920	1921–1930	1931–1940
Respiratory	45	45	25	55	28	29
Nervous	164	111	72	150	109	73
Alimentary	36	18	8	29	14	8
All other	46	42	24	51	45	20
Total	291	216	129	285	196	130

TABLE X.
The Deaths from Tuberculosis at Ages Ten to Fourteen Years Analysed by Form.

Form of Tuberculosis.	Males: Period			Females : Period		
	1911–1920	1921–1930	1931–1940	1911–1920	1921–1930	1931–1940
Respiatory	85	59	41	178	135	84
Nervous	77	70	51	85	63	34
Alimentary	22	16	11	26	15	8
All other	68	49	25	63	41	27
Total	252	194	128	352	254	153

Over the period 1908 to 1945, the mortality rates have fallen by some 60% in the case of respiratory tuberculosis, whereas they have fallen by about 95% for tuberculosis of the alimentary system. The fall in the tuberculosis rates for the central nervous system has been intermediate between these two and has amounted to about 75% of the 1908-1910 rate.

TABLE VIII.
The Mortality from Certain Forms of Tuberculosis in Early Childhood, the Mortality Rates being Calculated per Million per Annum for Children Under Five Years.

Form of the Disease.	Males : Period					Females : Period				
	1908–1910	1911–1920	1921–1930	1931–1940	1941–1945	1908–1910	1911–1920	1921–1930	1931–1940	1941–1945
Respiratory	79	49	29	27	32	69	44	29	24	19
Nervous	258	221	152	93	63	211	198	132	79	44
Alimentary	133	62	21	13	7	112	50	20	7	7
All other	63	55	42	22	25	27	40	37	22	25
Total	533	387	244	155	126	418	332	217	133	95

The rates for generalized forms, which we may assume were usually due to the human type, have only moderately declined, whereas the tuberculosis of "other systems", in which we have pooled lymph nodes, bone and joint, skin tuberculosis, *et cetera*, has declined considerably. This is perhaps due to the fact that tuberculosis of lymph nodes not included in the respiratory or gastro-intestinal system, such as the cervical nodes, was often due to the bovine type of bacillus in the earlier period. A similar sort of reasoning may be applied to the fall of the rates at ages five to nine and ten to fourteen years, but here we find that the differences are less pronounced. The general conclusion to be drawn is that over the period 1908 to 1945 hygienic measures against the bovine type of bacillus were more effective than those against the human type.

TABLE XI.

The Mortality Rates from Tuberculosis at Ages Five to Nine Years Analysed by Form. (Deaths per Annum per Million.)[1]

Form of Tuberculosis.	Males: Period			Females: Period		
	1911–1920	1921–1930	1931–1940	1911–1920	1921–1930	1931–1940
Respiratory ..	17	14	8	21	9	10
Nervous ..	62	36	24	57	36	25
Alimentary ..	14	6	2	11	5	3
All other.. ..	17	14	8	20	15	7
Total ..	111	70	43	109	65	44

[1] The column totals do not check exactly because of "rounding off"

TABLE XII.

The Mortality Rates from Tuberculosis at Ages Ten to Fourteen Years Analysed by Form. (Deaths per Annum per Million at Risk.)

Form of Tuberculosis.	Males: Period			Females: Period		
	1911–1920	1921–1930	1931–1940	1911–1920	1921–1930	1931–1940
Respiratory ..	36	20	13	75	46	27
Nervous ..	32	24	16	36	22	11
Alimentary ..	9	5	3	11	5	3
All other.. ..	29	17	9	27	14	9
Total ..	106	65	40	148	87	50

Tuberculosis in New South Wales.

J. B. Trivett (1909), Government Statistician in the State of New South Wales, reviewed the tuberculosis mortality and noticed a general tendency of the rates to fall over the years 1876 to 1908, especially in childhood. In Table XIII parts of his tables on his page 75 have been taken to show that the rates for "peritoneal"—that is, alimentary—tuberculosis had been very high, and that the reduction in the tuberculosis rates in early childhood between the periods 1883-1886 and 1905-1908 was chiefly due to a fall in the rates of the "peritoneal" forms, from 1940 to 250 for male and 1680 to 230 for female deaths per million *per annum*. The decline in the pulmonary forms over the corresponding period is much less pronounced. "Other forms" also have declined considerably.

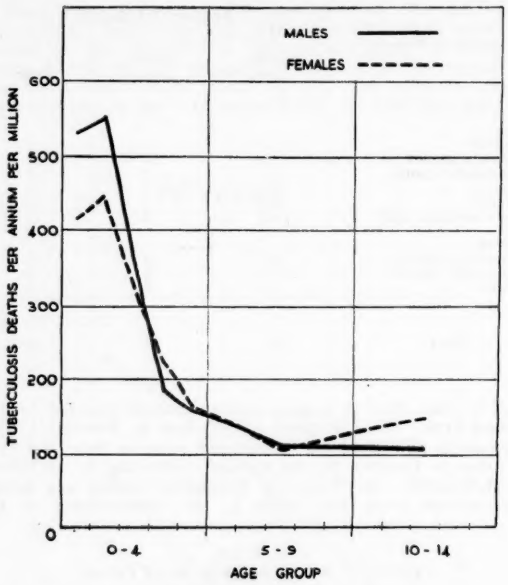

FIGURE II.

The death rates from tuberculosis of the sexes compared for the period 1911 to 1920. Arithmetic grid.

Summary.

1. The statistical data available on the tuberculosis mortality of childhood in Australia have been analysed.

2. A decline in the age-specific rates has been noted.

3. The decline in the mortality rates has been most pronounced in those systems where one can reasonably expect that the bovine type of bacillus was responsible for the majority of deaths in the earlier periods.

4. The effect of sex on the mortality rates has been noted. In the first few years of life the females have the more favourable rates; then for a few years the rates are approximately equal; whereas in the age group ten

TABLE XIII.

Annual Mortality Rates per Million in Each Age Group.
(From Trivett, 1909, page 75.)[1]

Age Group. (Years.)	Pulmonary Tuberculosis.		Peritoneal Tuberculosis.		Meningeal Tuberculosis.		Other Forms.		All Forms.	
	Male.	Female.	Male.	Female.	Male.	Female.	Male.	Female.	Male.	Female.
Period 1883–1886.										
0 to 4 ..	400	330	1940	1680	690	470	140	140	3170	2620
5 to 19 ..	220	340	—	—	10	20	30	30	260	390
Period 1905–1908.										
0 to 4 ..	110	100	250	230	260	230	*10*	*10*	*630*	*570*
5 to 19 ..	110	210	10	10	40	30	20	20	180	270

[1] Trivett's rates have been multiplied by 1000 to be comparable with the rates of the present paper.

to fourteen years the female rates exceed the male rates; this excess is almost entirely due to an increase of respiratory tuberculosis at this age.

5. A brief mention has been made of a publication by J. B. Trivett on the mortality in New South Wales, which shows that the decline in mortality observed over the period 1908 to 1945 had already begun in the latter part of the previous century, in New South Wales at any rate.

Acknowledgements.

I should like to thank Mr. D. W. Davies, of the School of Public Health and Tropical Medicine, Sydney, for the diagrams, and Dr. R. M. de Lambert for reading a draft of the paper and for helpful criticisms.

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THE USE OF "SODIUM PENTOTHAL" ADMINISTERED CONTINUOUSLY IN ABDOMINAL SURGERY.

By WIN FOWLES,
Brisbane.

IT was with some feeling of stepping into the unknown that in April, 1944, I commenced giving, for Dr. J. C. Bell Allen, "Sodium Pentothal" administered continuously as an anæsthetic agent when intraabdominal surgery was necessary. In my early days of so doing I gratefully accepted advice from Dr. J. J. C. Lamrock, of Sydney Hospital, Dr. A. Distin Morgan, of the Royal Alexandra Hospital for Children, and Dr. J. R. B. Beaumont, of the Royal South Sydney Hospital. They gave me knowledge of their experiences with this drug, for which I shall always be in their debt.

There was, and still is, a strong prejudice against its use in abdominal surgery on two main grounds: (*a*) its danger and (*b*) its power of relaxation.

In the past five and a half years I have averaged roughly five hundred abdominal anæsthetics per year using "Pentothal" alone or in conjunction with a small flow of intranasally administered oxygen. During that time most types of patients, from those with the most innocuous appendix to those with major conditions associated with advanced cardiac involvement, have been anæsthetized, and so far not one death in any way attributable to the anæsthetic agent has resulted.

One case that stands out in my mind was that of a woman, aged forty-seven years, who was undergoing hæmorrhoidectomy. As her anal sphincter was being dilated she developed a full laryngospasm, which was so persistent that she required intubation to relieve it. Several months later I was requested to give this same patient another anæsthetic for an appendicectomy. On this occasion, remembering my previous unhappy experience, I decided on the ethyl chloride-"open" ether sequence. The patient again had a violent laryngospasm, which in turn had to be relieved by intubation. She was a red-haired, extremely nervous woman and, in my opinion, any type of anæsthesia would have presented difficulties to the anæsthetist. It is well to remember, however, that "Sodium Pentothal" is a very powerful drug, and an incautiously administered anæsthetic in which it was used could well have fatal results.

On occasions when I am requested by a surgeon, who has not experienced its use in intraabdominal work beforehand, to give an anæsthetic for, say, a hysterectomy, the cry is raised: "But I must have full relaxation." The answer to that is, of course, to try it, and if the relaxation is not sufficient a change to some other type of anæsthetic can be made in the operating theatre. So far no surgeon has complained about the degree of relaxation obtained.

In considering the use of "Pentothal" for abdominal surgery, the type of patient, his or her physical condition at the time of operation and the type of operation to be performed must all be assessed by the anæsthetist.

The most difficult patients are those extremely nervous patients who, in any case, are a problem whatever the anæsthetic used. In bad cases of this type, this nervousness is manifest on occasions through the first ten to fifteen minutes of the operation when "Pentothal" is used. The plethoric, short-necked, bulbous-tongued patient is also one to be carefully watched, his difficulties mainly coming from a poor airway, which is usually best kept patent by a fairly wide-bore nasal catheter. The young and virile patient with heavy, well-conditionad abdominal musculature and a relatively high basal metabolic rate requires a far greater amount of the drug in proportion to his weight than does the debilitated patient, and, in my opinion, one gets a higher degree of toxicity and thus a lower margin of safety in these cases. For the advanced hypertensive "Pentothal" seems to be the anæsthetic of choice, and in the few of my own private cases of this type that I have been able to follow up, I have found a persistent fall in the patients' blood pressures, lasting in some cases over twelve months, so the anæsthetic would seem to have some therapeutic value. Patients with post-hæmorrhagic and anæmic states require a much smaller amount than those with a normal hæmoglobin value, and "Pentothal" in these cases should be given very slowly and with great caution if the safety margin is to be retained. Senility is not, in my opinion, a contraindication, and certainly for the debilitated patient "Pentothal" is the anæsthetic of choice.

In considering the use of "Pentothal" for intraabdominal surgery the site of the opening into the abdomen plays an important part. I have found it easier to maintain anæsthesia in operations on the lower part of the abdomen than in those on the upper part; and in operations requiring openings in the middle line than towards the flank. Thus in gynæcological operations, herniorrhaphy and appendicectomy full relaxation is usually relatively easy to maintain in the average patient. Operations in the epigastrium are, in the cases in which mid-line incisions are used, also satisfactory. Except for a very suitable patient I never use "Pentothal" alone for cholecystectomy.

Dr. John S. Lundy (1942), reporting its use in the Mayo Clinic between 1934 and 1940, states that in that period "Pentothal" was used in a total of 24,621 cases, of which 2245 involved intraabdominal conditions and 4439 gynæcological conditions.

Geoffrey Cottam (1948) reports its use in 7694 cases, of which 4656 involved major abdominal operations. He advocates a continuous flow of oxygen throughout the operation and the use of curare during the opening and closing of the peritoneum.

Surgeons and some of their appliances have their place in the scheme of things when the anæsthetist is considering using "Pentothal" for intraabdominal work. The surgeon who puts unnecessary tension on bowel and leaves large masses of bowel lying uncovered outside the abdomen is definitely contraindicated. Certain of the self-retaining retractors which undoubtedly would have made great appeal to the Inquisition have no place in association with "Pentothal" anæsthesia, in which any increased shock is important. I am certain that if some surgeons, using these instruments when only hand retraction is necessary, realized the damage they cause to the patients' pathological and physiological state by undue pressure, most of these appliances would find their rightful place in surgical museums.

Pre-operative measures in "Pentothal" anæsthesia are usually simple. The routine most generally used is "Nembutal" one and a half grains the previous evening about 9 p.m., and one hour before operation "Nembutal" three grains with atropine one one-hundred-and-fiftieth of a grain. I regard the smaller dose of atropine as important because it has been my experience that the incidence of laryngospasm has been reduced to about 25% of the figures I used to obtain when using one one-hundredth of a grain. For the extremely nervous, a combination of "Nembutal" one and a half grains, pethidine 50 milligrammes and atropine one one-hundred-and-fiftieth of a grain is used as an alternative one hour before operation. Morphine has a synergistic action with "Pentothal" as a respiratory depressant, and I therefore never use it.

Should a patient be unfortunate enough to possess a full set of artificial dentures, he or she usually arrives in the operating theatre *minus* these very useful articles. This habit probably originated in the days of Florence Nightingale and has persisted ever since. As for smooth anæsthesia under "Pentothal" an adequate mouth airway is essential, I invariably order the upper denture at least to be replaced before proceeding, thus preventing the collapse of the mandible and with it the movement of the tongue upwards and the cheeks of the patient inwards.

In the operating theatre I usually insist on a large, comfortable pillow, which completely removes any tension from the patient's larynx and trachea. My apparatus consists of a syringe holder with a clamp for attachment to the arm board, a 20 millilitre "Record" syringe, a tap fitting, a length of narrow tubing attached to a glass vein seeker, and a one and a quarter inch needle size 20, cut with a long bevel. As an adjunct I always carry a complete endotracheal apparatus, which fortunately I have had to use only once, in the case mentioned earlier in this article.

The patient's condition is then reassessed on the operating table as to the dose of "Pentothal" necessary. Some are grossly affected by the premedication, whilst others are not even slightly drowsy. For the average healthy patient I usually inject three to five millilitres of 5% solution rapidly and then wait ten to fifteen seconds to watch its effect. Individual responses to the drug are extremely variable, but full surgical anæsthesia is usually obtained with eight to fifteen millilitres in healthy patients. I recall one patient who required 36 millilitres before loss of consciousness, but who required only a further four millilitres for completion of his herniorrhaphy. On the other hand, for an anæmic, emaciated patient only seven millilitres were required for an appendicectomy. Maintenance during operation is assessed by the degree of relaxation, colour, respirations and observation of eye signs.

The patient's respirations are probably the most important single guide and in full surgical anæsthesia are at 10 to 12 per minute and shallow. When operative treatment is going to exceed thirty minutes I invariably insert a small-bore intranasal catheter about ten minutes after induction and use a flow of two to four litres of oxygen per minute through the tube. Throughout the operation the patient's colour should be kept within normal limits, and if cyanosis persists in the presence of oxygen then a change should be made to some other type of anæsthetic. This occurs only very rarely. I do not attach any importance to the pallor that occurs in certain patients, this being due to a mild peripheral ischæmia; in my opinion it in no way contraindicates the continued use of "Pentothal".

The eye signs used are the degree of dilatation of the pupil and, in the induction stage only, the corneal reflex. In full surgical anæsthesia the pupils are usually in mid-dilatation, but still react actively and rapidly to light. I consider that if this reaction to light is not immediate and pronounced, then the patient has had an overdose of the drug, and no more should be given until the light reflex is firmly reestablished. I use the corneal sign only if apnœa following induction lasts more than ten seconds. It gives only a very indifferent guide to the degree of anæsthesia.

The time taken to obtain full anæsthesia varies quite considerably with various types of patients, but with the average healthy type takes from thirty to one hundred and twenty seconds. With the lower abdominal operations the patient, even though quite deeply anæsthetized and completely relaxed, will quite often move an arm or a leg when the skin incision is made and also when the superficial veins are being tied off. If movement persists after this a dose of pethidine, 50 milligrammes given intravenously, usually suffices to end it. Pethidine forms a gel with "Pentothal", and should the same needle be used for its introduction then it should be cleared with a little sterile water, both before and after the pethidine is used. If this precaution is not taken a blocked needle will result.

From the commencement of the operation to its completion the patient must be closely watched and the depth of anæsthesia frequently checked. For the first twenty minutes or so of the operation maintenance doses are usually needed fairly frequently, and I usually give two to four millilitres of 5% solution each time according to the state of anæsthesia and the condition of the patient. The maintenance dosage after thirty minutes is usually very small, as the effects of continuous "Pentothal" administration are cumulative. With each successive injection of the solution the patient takes four or five deep breaths fairly rapidly before settling down to his normal anæsthetic rate. Respirations and colour are the cardinal signs during operation, and, as already stated, in procedures lasting more than thirty minutes I invariably use a small flow of oxygen through an intranasal catheter.

During the operative period the anæsthesia usually runs an uneventful course, except in those patients who have pronounced toxæmia as, for example, the patient with very acute appendicitis. In these cases excessive moisture is often present in the respiratory tract, and it is sometimes necessary to use a sucker to clear the pharynx. Should the pharynx be left uncleared then laryngospasm of varying severity will almost invariably result. This complication is, however, not common.

In those operations involving extensive handling of viscera, shock is sometimes a feature of the concluding stages. This has been especially noted in those patients who have mild toxic myocarditis following a condition of prolonged sepsis. "Coramine" one to two millilitres administered to these patients, and repeated in one hour if necessary, suffices to bring most back to normal.

In the immediate post-operative period rough handling and incorrect posturing are the chief causes of concern to the anæsthetist. I can recall one patient who had a perforated gastric ulcer being transported by hand in a sling up three narrow flights of stairs when a bed was available on the same floor as the operating theatre. Such actions as these are to be deplored, and, in my opinion, every hospital should be adequately equipped with trollies, trolly runways and, where necessary, lifts for the transportation of patients from theatre to ward.

As most people feel more comfortable, when asleep, using at least one full-sized pillow, I fail to see any reason why the anæsthetized patient, recovering from his anæsthetic, should not be thus provided. During recovery from "Pentothal" I regard this as especially important, as any extra tension on the trachea at this stage will tend to produce a blocked airway with its resultant dangers. Originally I relied on my instructions in the operating theatre to have this posturing done in the ward, but on so many occasions have I had to go to the ward to reestablish an airway in a very cyanosed patient that I make it a practice now to accompany the patient to the ward and see to the posturing personally. Since doing this I have never had to return to the ward for the purpose of reestablishing an airway. Use of a full-sized pillow with turning of the patient's head comfortably and without strain to one side, together with instructions to the ward attendants not to move the patient until he moves himself, results in a smooth recovery from the anæsthetic state with no discomfort to the patient attributable to the anæsthetic agent used.

Very rarely and at any time in the twenty-four hours' post-operative period, a patient may develop a condition which, for want of a better term, I call post-operative "Pentothal" intoxication. The symptoms consist of varying degrees of restlessness, together with delirium,

in extreme cases. The patient is liable to jump out of bed and rush round the ward objecting vocally and very strongly to any attempt at restraint. An injection of picrotoxin usually settles these patients down, and a feature of the condition is a complete amnesia of all events during the outburst. Although I have never seen it occur, the possibility of "burst abdomen", especially in those patients who have undergone one of the abdominal repairs, is exciting to those in charge of the patient who exhibits this condition. However, it is much more likely to cause a cerebral hæmorrhage in the attendants than any damage to the patient.

"Pentothal" anæsthesia for abdominal surgery is remarkable for the lack of post-operative discomfort following its use compared with most other anæsthetic agents. Nausea, vomiting and flatulence seldom occur, and when they do are almost invariably mild. Most patients who have had several experiences of anæsthetics will, after one experience of "Pentothal", usually insist on its use should any further surgery be required. They are mostly able to take nourishment by mouth immediately full consciousness is attained.

Conclusion.

These impressions of the use of "Pentothal" anæsthesia in abdominal surgery have been gathered by my own experiences, and the views expressed are largely my own. I have tried to set out in order the pitfalls of the various stages from consideration of the types of patient to full recovery in the ward. Some of my opinions are undoubtedly controversial, but collectively they indicate the means of administering a safe and effective anæsthetic with "Pentothal" for abdominal operations.

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Reports of Cases.

DOUBLE UTERUS WITH COMPLETE VAGINAL SEPTUM.

By PHILIP C. THOMAS, M.R.C.O.G.,
Perth.

WHEN the Müllerian ducts fail to fuse, various types of uterine abnormality are produced, the level at which the fusion stops determining the type and degree of deformity. Stanley Way (1945), reporting on 18,000 obstetrical hospital admissions and 10,000 gynæcological admissions in Newcastle, England, over a seven-year period from 1938 to 1945, found 23 cases of this type of uterine abnormality. The condition in the case being reported here appears to be either a *uterus bicorpus bicollis* or else a *uterus didelphys*, apparently the only difference between the two being in the depth of the cleft between the two uterine halves. Without laparotomy one was unable to be more definite than this, although bimanual vaginal examination and the hysterograms rather favour a diagnosis of *uterus didelphys* (Figures I and II).

Clinical Record.

The patient, a spinster, aged forty years, sought medical attention in August, 1948, on account of severe incapacitating dysmenorrhœa. The menarche had occurred at the age of seventeen years and the menstrual periods came regularly every twenty-eight days and lasted for three days with small loss. The pain lasted throughout the entire period, was mainly located in the right iliac fossa, and was severe enough to warrant her going to bed for three days. Fourteen years earlier she had had a dilatation of the cervix and curettage, from which she had derived slight temporary relief. The genital tract abnormality had passed unnoticed at that time.

Upon examination of the vulva, a septum was visible at the introitus, which on digital exploration was found to divide the vagina into two lateral components with

FIGURE I.
Uterus bicorpus bicollis.

FIGURE II.
Uterus didelphys. Cleft between the two uteri reaches to the cervico-vaginal junction.

a small cervix palpable in each vaginal vault. When the patient was examined *per vaginam* under anæsthesia it was thought that the abnormality was of the didelphys type. A right hysterogram was prepared which showed one half (Figure III), and then, with the right cannula still

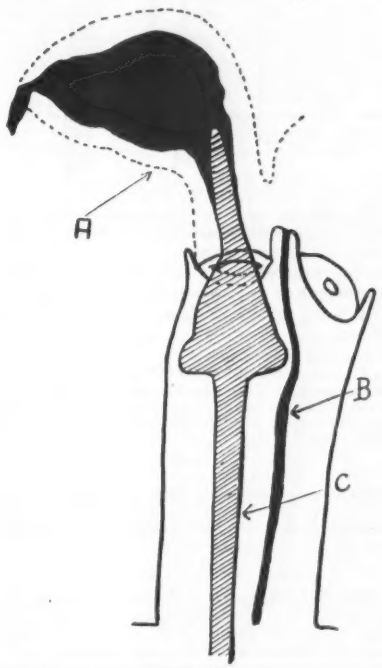

FIGURE III.
Right hysterogram. A: suggested outline of uterus (right); B: vaginal septum; C: uterine cannula.

in situ to prevent escape of oil, a hysterogram was prepared on the left side and a second film taken (Figure IV). The films showed that each cornu communicated with a patent Fallopian tube and suggested that the right section was the larger of the two.

In view of the not uncommon coexistence of congenital anomalies of the genital and urinary tracts, a complete urological survey was made, but no abnormality was found.

Comment.

It is possible that in this case the presenting symptom, namely dysmenorrhœa, may have been due to some incoordination of function and muscle action between the two halves of the double uterus.

This patient has never been interested in matrimony or sex life, and so, for the moment at any rate, there is no

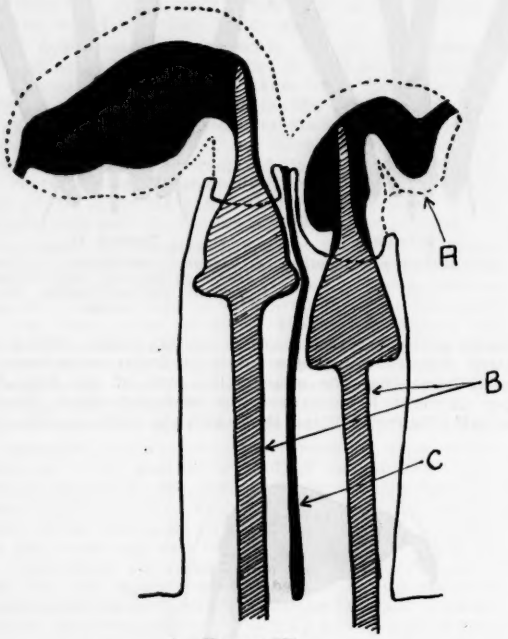

FIGURE IV.

Bilateral hysterogram. A: suggested outline of uterus (left); B: uterine cannulæ; C: vaginal septum.

indication for any surgical treatment on account of the uterine abnormality *per se*, unless it becomes necessary because of her dysmenorrhœa, by far her main preoccupation.

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AGRANULOCYTOPENIA FOLLOWING "CHLOROMYCETIN": REPORT ON TWO CASES.

By P. F. GILL, M.B., B.S.,
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RECENTLY the new antibiotic "Chloromycetin" has become available in Australia. At present its prohibitive price is a strong deterrent against its widespread use in general practice, but as with the earlier antibiotics this no doubt will soon fall to a suitable level. Then the following experiences may be of common occurrence or may be forgotten as rarities. The purpose of this article is not to write a treatise on "Chloromycetin"—for I know nothing about its chemistry, mode of action *et cetera*—but to point out that the drug can be dangerous and it should therefore be treated with a little more respect than the current literature would lead us to believe.

Case I.

A baby girl, aged thirteen months, was admitted to the hospital on January 21, 1950, suffering from pink disease of four months' duration. She had been treated along the usual lines at home with little change either way. Two weeks before her admission to hospital she became pyrexial and rapidly lost ground.

On her admission to hospital she showed the typical features of pink disease, and a crop of pustules on the head and face was held responsible for the pyrexia, which disappeared after a few days' local and parenteral treatment with penicillin. A routine blood count on her admission to hospital showed a normal picture. With the clearing of the skin condition she began to improve somewhat. The publication of the work of Cheek and Hicks (1950) on the nature of pink disease prompted further investigation and treatment. A blood count on February 3 gave the following information. The erythrocytes numbered 5,300,000 per cubic millimetre, the hæmoglobin value was 106%, and the colour index was 1·0. The leucocytes numbered 11,000 per cubic millimetre, 42% being neutrophile cells and 58% lymphocytes. The platelets were normal, and the hæmatocrit was 50%.

The patient was therefore started on salt therapy, one and a half teaspoonfuls daily, and desoxycorticosterone acetate, four milligrammes daily. The latter was unprocurable for several days, and it was not until February 8 that she received it. Just after the commencement of this therapy she began to have frequent loose blood-stained offensive bowel motions, and in twenty-four hours was dehydrated. The fluid balance was restored by the transfusion of 200 millilitres of blood, followed by 500 millilitres of normal saline, given intravenously, but she continued to have loose frequent motions. Salmonella group C was grown on culture from the fæces. At this stage she was clearly a candidate for "Chloromycetin" therapy, which was commenced on February 4. She received 250 milligrammes (chloramaphenicol—Parke, Davis) *statim* and 100 milligrammes at each feed (eight feeds a day). The drug was given mixed in the food, but for the first few doses she vomited some of the food, so that feeding by nasal catheter was instituted. Later she had oral feedings without ill effects. The course was to last for ten days. Regular daily cultural examination of the stools was made with the following interesting results. On February 4, the day of commencement of treatment, "many non-lactose fermenting colonies" were reported to be present; on February 5 "few non-lactose fermenting colonies" were present; from February 6 to 12 no colonies were found.

Under this regime the improvement was spectacular, and by the second day her motions were back to normal. She lost her irritability and even later began to smile. On February 11, as a guide to the effectiveness of the salt and desoxycorticosterone acetate therapy, a further blood count was made, with the following alarming result: the erythrocytes numbered 5,300,000 per cubic millimetre, the hæmoglobin value was 100% and the colour index was 1·0; the leucocytes numbered 10,200 per cubic millimetre, 9% being neutrophile cells, 88% lymphocytes, and 3% eosinophile cells; the platelets were normal and the hæmatocrit was 50%.

Both desoxycorticosterone acetate and "Chloromycetin" were stopped immediately. She received a further transfusion of 200 millilitres of fresh blood, and was given 50 milligrammes of pyridoxin and penicillin as a prophylactic measure daily. She remained in good health, and her blood picture rapidly returned to normal, as follows: On February 14 the leucocytes numbered 9700 per cubic millimetre, 35% being neutrophile cells, 63% lymphocytes and 2% eosinophile cells. On February 16 the leucocytes numbered 9400 per cubic millimetre, 40% being neutrophile cells, 58% lymphocytes and 2% eosinophile cells.

The administration of pyridoxin was now stopped and that of desoxycorticosterone reinstituted. Regular leucocyte counts have shown no further tendencies to agranulocytosis, so I can only assume that the "Chloromycetin" was responsible.

Case II.

This case is even more interesting, for in view of the previous case leucocyte counts were made daily. A baby boy, aged four months, was admitted to hospital on February 13, 1950, with a history of increasing diarrhœa of two weeks' duration, with blood and slime in the motions. He was not particularly ill on his admission to hospital, but his bowel action showed no signs of returning to normal on routine gastro-enteritis treatment. The result of the bowel swab revealed a salmonella infection, and he was recommended for "Chloromycetin" therapy.

On his admission to hospital a blood count gave the following information: the erythrocytes numbered 3,400,000 per cubic millimetre and the hæmoglobin value was 66%; the leucocytes numbered 5000 per cubic millimetre, 46% being neutrophile cells, 49% lymphocytes and 2% eosinophile cells.

After one day's therapy, as outlined previously, the leucocytes numbered 10,600 per cubic millimetre, 59% being lymphocytes, 33% neutrophile cells, 5% monocytes and 1% eosinophile cells. The trend towards agranulocytosis was even more obvious the next day; the leucocytes numbered 7000 per cubic millimetre, 23% being neutrophile cells, 76% lymphocytes and 1% eosinophile cells.

Administration of the drug was stopped immediately, and the routine leucocyte counts made over the next few days showed a return to normal. In passing, the child's condition benefited greatly by only two days therapy, and he has remained well.

Comment.

As far as I can ascertain, these are the only cases so far reported of toxic effects of the drug on the bone marrow. The dosage given was along the lines of that suggested by the literature. The first patient received 6·85 grammes; the second patient received only 1·75 grammes. In the few reports on the drug in the literature that I have been able to review, the outstanding feature is the wide variation in dosage with apparent equally good results.

In a series of Indian cases of typhoid fever treated with relatively small doses (because of the scarcity and high price of the drug), Vakil suggests that it might have a tendency to cause leucopenia, but most other series have taken large doses without toxic effects. Comparatively large amounts were given to infants during an outbreak of gastro-enteritis at the Birmingham Children's Hospital late last year, and none showed toxic phenomena.

It seems that the dosage cannot readily be incriminated, so the explanation must fall back on to personal idiosyncrasy. Whatever the explanation, "Chloromycetin" apparently can cause agranulocytopenia.

Acknowledgements.

I wish to thank Dr. P. A. Earnshaw and the Sisters of Mercy for their permission to publish these histories.

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Reviews.

THE SCIENCE OF VITAMINS.

IN 1921, Dr. Eddy produced the first edition of his vitamin manual. We now have his "Vitaminology" of 1949.[1] During his lifetime the author, who is Emeritus Professor of Biochemistry, Teachers' College, Columbia University, has witnessed and been an active participant in the phenomenal growth of knowledge of vitamins. Each of the vitamins now has an extensive and often bewildering literature. Eddy has very ably reviewed the salient facts of this literature in the case of each vitamin and has encompassed these facts within a single volume. In his introduction he states that "an attempt has been made to stress what functions appear established, what have been suggested and what remain for further investigation". These important points are not always as clearly defined in nutrition literature as they should be, but Eddy has succeeded in clearly delineating the established from the suggested facts. The subject of each vitamin is discussed widely. Aspects of biochemistry, physiology, pathology, public health nutrition, animal nutrition *et cetera* are presented under easily referable headings. Particular attention has been paid to the presentation of the structural formulæ of the natural and synthetic vitamin compounds where these are known, with a discussion of the nature of the chemical actions in which they are known or thought to participate. As can be inferred from the title, there is no attempt made to cover the subjects of any nutrients other than the vitamins.

On reading this book one is impressed not by how much is known about the vitamins and their actions, but by how much remains for elucidation. The bibliographies are well selected to lead the student to the problems.

We confidently recommend the book to all who wish to have an up-to-date treatise on vitamins in a single volume. It is a useful reference monograph, in our opinion the best we have seen, for physicians studying for higher degrees, and for those engaged in teaching and research in biochemistry or in human and animal nutrition.

[1] "Vitaminology: The Chemistry and Function of the Vitamins", by Walter H. Eddy, Ph.D.; 1949. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson. 9" × 6¼", pp. 380. Price: £3 4s. 6d.

BRAIN TUMOURS AND NEUROSURGERY.

ERNEST SACHS, who has long been regarded as one of America's most experienced neurosurgeons, has now revised his earlier edition of "The Diagnosis and Treatment of Brain Tumours" and incorporated with it, as a second edition, his more recent book, "The Care of the Neurosurgical Patient".[1] Some rearrangement of the subject matter has taken place, and some of the chapters have been intermingled, to form a very informative yet compact book, covering most aspects of neurosurgical diagnosis and treatment. Intended primarily for medical students and house officers, the significant features are emphasized by quotations from detailed case histories, especially those illustrating common errors; these show how pitfalls may be avoided.

Firm in the faith that all neurological surgeons must have a thorough training in neurology and not merely be the hands that work for the medical neurologist, Dr. Sachs devotes the first chapter to the surgical anatomy and physiology of the nervous system, made the more interesting by the help received in its preparation from Professor S. W. Ranson.

The essentials in both these fields are covered fully and clearly with the help of over 100 references. Many of these published between 1885 and 1921, and quoted as if they were new, might now be discarded. Cushing's transverse skin incision for decompression is never heard of now; and the confusion which previously may have occurred from the use of the words "hemianopsia" and "hemianopia" to describe losses in visual fields no longer exists, as in all literature, "hemianopia" has been used for years in references to the loss of half the visual field. In the discussion on the disturbances of speech which occur with cerebral lesions, no reference is made to Nielson's book on "Aphasia, Agnosia and Apraxia", which is a complete treatise on this complicated neurological problem; and, in the discussion on the localization of function in the cerebrum, no mention is made of Penfield's extensive experimental studies in man.

The second chapter is concerned with the various methods of examination. In most instances, the methods advocated are simple and standardized, but in other neurosurgical clinics, the Bjerrum screen is preferred to any form of small perimeter for routine estimation of the visual fields for quantitative and qualitative defects. A large series of skiagrams is reproduced to illustrate various cranial and intracranial lesions, but in some of them the lesion, especially some of the areas of calcification, are hard to see. Paget's disease of the skull is common enough to warrant inclusion.

[1] "Diagnosis and Treatment of Brain Tumours and Care of the Neurosurgical Patient", by Ernest Sachs, A.B., M.D.; Second Edition; 1949. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 9¼" × 6", pp. 554, with 348 illustrations. Price: £7 17s. 6d.

The author advocates and acclaims Dandy's method of ventriculography as the best means of localizing brain tumours and supports this contention with a series of characteristic films and case histories; but no details about the performance of pneumoencephalography are given and no pneumoencephalograms are shown at all.

Similarly, electroencephalography and cerebral angiography are dismissed in very brief fashion. No tracings or films are shown of normalities or of the characteristic abnormalities which can be diagnosed by these methods, so well perfected in recent years, that, in many neurosurgical clinics, ventriculography has almost been given up, and the reader is left to obtain the necessary information about these methods from references given as footnotes.

Following a clear and concise chapter on the pathology of brain tumours with many excellent photomicrographs and illustrations of vascular abnormalities, the general symptoms and signs of increased intracranial pressure are discussed. Many tables dealing with the frequency of convulsions in the various types and situations of tumours are no longer of any great interest and could be well replaced by conclusions only.

The focal symptoms and signs of intracranial tumours are dealt with in the next two chapters, and all the salient and important features of each type of tumour are illustrated by full clinical and operative notes of typical cases.

Aphasia is discussed according to Head's classification of verbal, syntactical, nominal and semantic varieties. The lengthy quotation of Head's own description of the last two types is unnecessary and too confusing for students. The same applies to the quotation of Foster Kennedy's description of dreamy states.

In all cases of glioma, it is rightly recommended that an attempt be made to remove them completely by lobectomy or at least partially, since the advent of better electrosurgical apparatus and of hæmostatics such as gelfoam has made the control of hæmorrhage easier.

The syndromes of cerebellar and posterior fossa tumours are particularly well described and illustrated by case notes, but no reference has been made to Torkildsen's palliative operation of "short-circuiting" the aqueduct of Sylvius in cases of obstructive hydrocephalus due to tumours of the third ventricle, the brain stem and pineal region, a procedure which has proved successful with many of these lesions, which have a low grade of malignancy.

The pituitary gland has a chapter to itself in which are described its various diseases and tumours, and their management from endocrine, radiotherapeutic and surgical points of view. Several charts of the visual fields in cases of pituitary lesions are included, but none to illustrate the classical finding of bitemporal hemianopia.

In the last chapter of the original first part of the book, the differential diagnosis between brain tumours and many other conditions is ably and concisely discussed. Particular attention is given to brain abscesses and their treatment which has been completely changed with the advent of the antibiotics. Whilst agreeing that complete excision is the ideal method of dealing with a chronic well-encapsulated abscess, we think that there is still a place for "repeated aspirations" of acute and subacute abscesses, followed by instillations of an antibiotic, to which the infecting organism is sensitive. Mention might have been made here of the value of instilling a small amount of "Thorotrast" after the first aspiration in order to outline the abscess cavity in subsequent skiagrams (pyography) and help to "fix" its capsule.

Most of the other types of intracranial infections, such as encephalitis, tuberculous meningitis and syphilis, are discussed, but in future editions, lead encephalopathy, especially in children, and torulosis might well receive some consideration. Other conditions briefly mentioned are aneurysms, arteriosclerosis, metastatic tumours and multiple sclerosis.

For anæsthesia, the author is a strong advocate of local infiltration with weak solutions of "Novocain" without any premedication to stupefy the patient. In lengthy major operations he employs "Avertin", a practice which most neurosurgeons follow today. His operative methods and details of neurosurgical technique and instruments, used in all the different approaches, are clearly described and well illustrated. The many tips given for the control of hæmorrhage and the avoidance of post-operative clot are excellent, and obviously the result of long personal experience; but there does not seem to be any adequate reason for stating that "a cerebellar wound should never be drained". In a description on the ways of dealing with a cranio-pharyngiomatous cyst, reference could also have been made to the method advocated by Scarff whereby, after the solid portions and debris of the cyst have been removed by suction, the cavity is made to communicate with one of the lateral ventricles so that its secretions can pass out with the cerebro-spinal fluid and be absorbed.

If abscesses are dealt with correctly nowadays cerebral fungi should not occur and there should not be any necessity to issue a warning against shaving them off!

In operations for trigeminal neuralgia, instead of the classical extradural approach via the temporal fossa, originally credited to Frazier, the intradural route, advocated by Wilkins, has now been adopted with gratifying results because a differential sensory root section can be more easily accomplished and the motor root more often saved.

In a short account of the treatment of communicating hydrocephalus, the technique of coagulation of the chorioid plexus is described and the important formula for the replacement fluid is given. In advanced cases, uretero-spinal anastomosis is advocated and described, a method which has been simplified recently by the use of fine polythene tubing. Penetrating wounds and compound fractures of the skull, cranioplasty and subpial resections of cortex for epilepsy are also dealt with. In the last-named condition, the author recommends use of the galvanic current as a stimulator rather than the faradic, which may start severe convulsions.

The technique of laminectomy for the removal of all forms of intraspinal tumours and herniated intervertebral disks is well demonstrated. In cases of *spina bifida*, Penfield's theory that the sac should be preserved to prevent hydrocephalus is not agreed with. Exact details of suturing severed nerves, replacing bone flaps and closing wounds are shown; and the importance of the correct application of dressings and bandages is stressed, points which might well be taken to heart by present-day resident medical officers and nurses, not only in neurosurgery. Many important post-operative instructions are laid down, among them being an injunction not to be too proud to make a rectal examination and remove hard scybalæ from patients to whom several enemata have been "given without any result". Sutures are left in the skin for only one day, to avoid unsightly scars, yet if this were done routinely, it is felt some reactionary hæmorrhages would occur.

In a short concluding chapter, the author ably reviews the development of neurosurgery and its accomplishments and makes a plea for its continuance as a specialty as distinct from general surgery and medical neurology.

When the general soundness of this book and its careful attention to detail are taken into consideration, the above-mentioned criticisms are relatively minor. It is beautifully set out and printed, and should be read by all those interested in neurosurgery.

THE PROGRESS OF SURGICAL EDUCATION IN IRELAND.

IT is barely a century since the people of Great Britain took legislative action to ensure that medical services in future should be properly organized, adequately controlled, and provided by a profession with the necessary education, training and qualifications to maintain a high standard of practice. Even in the Middle Ages there was some semblance of stability, uniformity and direction in the Church, the Law and in the various crafts, which were imbued with the corporate spirit by reason of membership in the respective guilds. Five hundred years ago English physicians sought to have the practice of medicine regulated and controlled by Act of Parliament, but their petition was ignored by the legislators and nothing further was done. Until the passing of the *Medical Act* of 1858, the medical profession had to rest content with spasmodic legislation, the inroads of unrestrained charlatanism and the continued ignorance and incompetence of many licensed practitioners. Isolated attempts were made in the eighteenth century to raise the standard of medical and surgical practice, and one interesting facet of this praiseworthy movement is clearly shown in "An Account of the Schools of Surgery, Royal College of Surgeons, Dublin (1789-1948)",[1] by Dr. J. D. H. Widdess, librarian and lecturer in biology in that College, which is still following the tradition handed down from its early founders and benefactors "to establish a liberal and extensive system of surgical education" in Ireland.

In 1780 the Dublin surgeons made the first move to dissociate themselves from the barbers in what was termed a "proposterous [*sic*] and disgraceful union", and they endeavoured to obtain a Royal Charter that would

[1] "An Account of the Schools of Surgery: Royal College of Surgeons, Dublin, 1789-1948", by J. D. H. Widdess, M.A. (Dublin), L.R.C.P. and S.I.; with a foreword by William Doolin, F.R.C.S.I.; 1949. Edinburgh: E. and S. Livingstone, Limited. 9¾" × 6¼", pp. 124, with 16 illustrations. Price: 17s. 6d.

incorporate them in a scientific body for the advancement of the profession by the cultivation and diffusion of surgical knowledge". The College duly received its Charter in 1784, and the first students were required to pass an entrance examination in Latin and Greek classics, and, if approved by the president and two censors, they were apprenticed to a member of the College, were permitted to attend lectures in anatomy, physiology and pharmacy, and, if successful in the final examination, were awarded the letters testimonial. In 1789 the Dublin surgeons were able to establish chairs of anatomy, physiology, surgery, midwifery and pharmacy in a school under their own direction. This was the first step taken in Great Britain and Ireland towards the systematic education and training of apprentices, who were to form the vanguard of a competent body of general practitioners whose diploma was universally accepted at least fifty years before the triple qualification became a legal requirement.

Certain events connected with the history of the Dublin College of Surgeons are not without their tinge of humour. Professor William Lawless, who occupied the chair of anatomy from 1794, was expelled by the governing council four years later for his active participation in underground politics. He was by no means alone among his professorial colleagues in his sympathies with the cause of the United Irishmen, but the rest of the teaching staff showed more discretion. Then there are amusing anecdotes concerned with the need to procure bodies for dissection, and the peculiar situation that was common to all schools of anatomy before the *Anatomy Act* of 1832 is well illustrated by two interesting plates showing a grave in one of the Dublin churchyards completely encaged behind iron railings to protect it from the "resurrection men", and a watch-tower of strong masonry in another Dublin churchyard, where relatives kept night vigils so long as a deterrent was necessary to warn off intending body-snatchers.

The names of several Dublin physicians and surgeons, who were closely connected with the early days of the Royal College of Surgeons in Ireland, are still familiar to us in modern medical literature, Abraham Colles, John Cheyne, William and Whitley Stokes, Robert Adams and John Houston. They all had the same idea: that there could be no distinction between "physic" and surgery and that it was impossible to draw any line of demarcation between them. As a distinguished Dublin teacher wrote in 1841: "It is certain that there cannot be a good physician who has not the knowledge of a surgeon, or a good surgeon who has not the knowledge of a physician; therefore it is obvious that both should be educated alike."

CLINICAL SURGERY.

"AN INTRODUCTION TO CLINICAL SURGERY", by Charles F. Saint, formerly professor of surgery, University of Cape Town, has reached its second edition.[1] The only change is the inclusion of numerous clinical photographs and diagrams designed further to illustrate the text.

This work, which is essentially clinical, is packed with experiences in observation and diagnosis gleaned from a lifetime of surgical teaching and surgical practice. Its theme as stated by the author is "A reasoned explanation of surgical note-taking".

Some notion of the scope of the book may be gained from the titles of some of the various schemes of note-taking. These include injuries, inflammation, tumours, abdominal emergencies, stomach and duodenum, appendicitis, rectal cases, herniæ, scrotal swellings, calculi, swellings of the breast, fractures, jaundice, swellings of the neck and such-like subjects. Some idea of the way the author handles these subjects may be obtained by a detailed survey of some of the schemes.

Under the head of stomach and duodenum, he gives reasoned explanations for the variations in the type of pain which occurs in disease in these regions—for the changes in its mode of onset, its character, its severity, and its distribution. And in the case of other symptoms which are not so constant, he analyses the mechanism of their origin and describes the significance of their presence or absence. Then follows a rather meagre account of methods of physical examination and the inferences that may be drawn from these. "Adnexed" is a collection of photographs of pathological specimens and of radiographs of diseased conditions. Little attempt, however, has been made to use these examples of dead and living pathological conditions to confirm the hypotheses in the author's "reasoned explanations".

In the scheme which has to do with acute appendicitis, it is hard to agree with all the explanations offered for the variations in the pain of acute appendicitis; that is, for the variations in its mode of onset, its situation, its severity, and its association with vomiting. And while it may be possible to agree with the author's hypothesis for the association of dyspepsia with chronic appendicitis, it is not so easy to accept his statement that it follows from these reasons that it is necessary, in many cases, to combine a pyloroplasty with an appendicectomy in order to effect a complete cure of the dyspepsia.

Many of the author's reasoned explanations may seem somewhat far-fetched; it would be too much to expect that they could all be sound. Nevertheless they are the product of great experience combined with much thought, and must encourage the inquiring type of mind; they will put some sort of structure into the accumulation of clinical knowledge; and also they will serve to align the clinical side in diagnosis in proper perspective.

The book will be found most useful to the student and the general practitioner.

[1] "An Introduction to Clinical Surgery: Surgical Wherefores and Therefores: A Reasoned Explanation of Surgical Note-Taking", by Charles F. M. Saint, C.B.E., M.D., M.S., F.R.C.S. (England), Hon. F.R.A.C.S., Hon. F.G.S.S.; Second Edition; 1949. Cape Town and Johannesburg: Juta and Company, Limited. 9¼" × 6", pp. 396, with 372 illustrations. Price: 45s.

A BIOGRAPHICAL HISTORY OF TUBERCULOSIS.

IT is not surprising to learn that Professor S. Lyle Cummins has been able to advance the knowledge of his own specialty when we find that he is so familiar with the works of his illustrious predecessors. He has made a profound study of their writings from the early seventeenth century to the present day, and the result of his labours was presented in several papers before the Section of the History of Medicine of the Royal Society of Medicine in London. These essays combined with much additional material have now been collected together in the form of a book, "Tuberculosis in History", and as such they are a valuable contribution to the literature of this subject.[1]

The contents are divided into three main parts, each one containing a number of biographical sketches of medical practitioners who in their day made some definite contribution to the knowledge of this persistently prevalent, perplexing and pernicious disease. The first part is given entirely to the British school of doctors, including Christopher Bennet, Thomas Willis, Thomas Sydenham, Richard Morton, Benjamin Marten and George Bodington. The second part comes under the heading of "Early Continental Phthisiologists" and contains interesting accounts of the life and work of Auenbrugger, Corvisart, Bayle, Laennec, Piorry and, finally, Jean Antoine Villemin, who was the first to prove scientifically that tuberculosis must be looked upon as an infectious disease. The last part brings the story to a satisfying conclusion with a vivid pen picture of "two remarkable figures in the history of tuberculosis", Edward Livingston Trudeau and Robert Koch, which leaves us upon the threshold of the modern era and with many problems still unsolved.

It is soon plainly discerned that Professor Cummins has probed deeply into the original writings of the medical pioneers whom he has selected to provide a basis for the full development of the main theme. Furthermore, his own expert knowledge and experience as a specialist in the treatment of the disease have enabled him to supply a running commentary throughout the entire text, thus making the older writings more intelligible to the reader. At the same time he has ingeniously sifted out from the wordy literature of the past all those original ideas which are now recognized as representing a forward movement towards a better understanding of pulmonary tuberculosis.

This book should be read by every practising physician, as the subject matter not only encompasses the history of a disease, but deals interestingly with the evolution of modern methods of physical diagnosis which are always in danger of being overshadowed by later inventions. Another agreeable feature is the inclusion of several sepia-tone portraits, and the picture of Thomas Sydenham by Mary Beale is much to be admired. Certain typographical errors will be doubtless eliminated in subsequent editions, such as occur more than once in the names of Cohnheim, Burdon-Sanderson and Van Swieten, who, by the way, was not in

[1] "Tuberculosis in History: From the 17th Century to our own Times", by Professor S. Lyle Cummins, C.B., C.M.G., LL.D., M.D., with an introduction by Sir Arthur Salisbury MacNalty, K.C.B., M.D., F.R.C.P., F.R.C.S.; 1949. London: Baillière, Tindall and Cox. 8½" × 5¼", pp. 225, with 12 illustrations. Price: 21s.

actual fact "attracted from Leyden to the Vienna School". He left Leyden because religious disabilities precluded his elevation to a professorial chair, and proceeding to the Austrian capital with encouragement from the Empress Maria Theresa, he set about laying the foundations of a medical tradition to be hailed by posterity as the Old Vienna School.

DISEASES OF WOMEN.

THE eighth edition of "Diseases of Women", by ten teachers, has been published.[1] Since the appearance of the last edition, Messrs. Arthure Bell and Roques have replaced as authors Mr. Victor Bonney, Mr. Goodwin and the late Sir Comyns Berkeley. The general design of this excellent book remains the same, but much of the text has been revised and rewritten and some new illustrations have been added.

The sections on the anatomy of the pelvic organs are well arranged and the illustrations and drawings are clear and plentiful. The section on the physiological action of the endocrine glands has been entirely rewritten and brought up to date. The views expressed on endocrinology conform with the opinions and teachings of the British school of gynæcology and the dangers of the indiscriminate use of the sex hormones are stressed. The authors point out that the clinical application of sex endocrinology has been disappointing and that our knowledge of the human endocrine mechanism lags behind that of experimental work on animals.

The various other sections are dealt with clearly and concisely and brought up to date.

The only adverse criticism of this book is in regard to the section on surgical procedures. From the student's point of view, this section will require supplementing and it is a pity that more space and detail have not been given to this important section. A few more diagrams and drawings could have been used to advantage and this could have been done, without an increase in the size of the book, by a condensation of the rather long section on neurasthenia and neurosis. This subject is adequately covered in the larger medical text-books.

This book, which has been a standard gynæcological text-book for thirty years, can be recommended to students and practitioners as a concise, orthodox and well-written book on up-to-date gynæcological teaching.

FIRST-AID MANUAL.

THE ninth edition of the "British Red Cross Society First Aid Manual" by Sir Harold E. Whittingham and Sir Stanford Cade now appears after a gap of five years since the last edition, this hiatus being due mainly to the war.[2] The work has been completely revised and many of the chapters have been entirely rewritten in the light of knowledge gained from the treatment of war-time casualties. This is perhaps best seen in the chapter on shock, in which a brief but complete account of the modern concept of the subject is clearly given. In regard to the important subject of artificial respiration, both Schafer's and Eve's methods are described, but no mention is made of the use of carbon dioxide as a respiratory stimulant in cases of asphyxia or drowning; however, this measure may perhaps be considered beyond the scope of first-aiders even though in this country most ambulances carry the "Carbogen" outfits and frequently make good use of them. The first-aid treatment of all types of injuries from burns and poisonings to fractures and dislocations is covered. As well as being of great use to all students of the subject, the book will be invaluable as a guide for doctors and ambulance men who may have to instruct laymen in the rudiments of first aid to the injured. Being modestly priced, this manual should be in the hands of all those concerned with the subject whether in the home or in the factory, for with this book as a handy reference emergency treatment is made efficient and as easy as possible.

[1] "Diseases of Women", by ten teachers under the direction of Clifford White, M.D., B.S. (London), F.R.C.P. (London), F.R.C.S. (England), F.R.C.O.G., edited by Clifford White, Frank Cook and Sir William Gilliatt; Eighth Edition; 1949. London: Edward Arnold and Company. 8¼" × 5¼", pp. 476, with 170 illustrations. Price: 25s.

[2] "British Red Cross Society First Aid Manual No. 1", by Sir Harold E. Whittingham, K.C.B., K.B.E., F.R.C.P., and Sir Stanford Cade, K.B.E., C.B., F.R.C.S.; Ninth Edition; 1949. London: MacMillan and Company, Limited. 6" × 5¼", pp. 322, with 171 illustrations. Price: 3s.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Emergency Surgery", by Hamilton Bailey, F.R.C.S. (England), F.A.C.S., F.I.C.S., F.R.S.E., assisted by Norman M. Matheson, M.B., Ch.B., M.R.C.P. (London), F.R.C.S. (England), F.A.C.S.; Part III; Sixth Edition; 1950. Bristol: John Wright and Sons, Limited. London: Simkin Marshall, Limited. 10" × 6¾", pp. 167, with 246 illustrations, some of them coloured. Price: 21s. per part.

Part II of this work was reviewed in this journal in November, 1948.

"The Rheumatic Diseases", by G. D. Kersley, M.A., M.D. (Cantab.), F.R.C.P. (London), T.D.; with a foreword by Sir Francis R. Fraser, M.A., M.D. (Edinburgh), F.R.C.P. (London); Third Edition; 1950. 8" × 5", pp. 164, with illustrations. Price: 15s.

An attempt to give a general outline of rheumatic diseases in as short a space as possible.

"Introduction to Psychiatric Nursing", by Marion E. Kalkman, R.N.; 1950. New York, Toronto and London: McGraw-Hill Book Company, Incorporated. 9" × 6", pp. 356, with 13 illustrations. Price: $3.75.

Written for those commencing psychiatric nursing that they may learn that their work is not purely custodial.

"The 1949 Year Book of Drug Therapy (November, 1948-October, 1949)", edited by Harry Beckman, M.D.; 1950. Chicago: The Year Book Publishers, Incorporated. 7" × 5", pp. 724, with 133 illustrations. Price: $4.75.

A new member of the Practical Medicine Series of Year Books.

"Regional Ileitis", by Burrill B. Crohn, M.D.; 1949. London: Staples Press, Limited. 8½" × 5½", pp. 244, with 74 illustrations. Price: 30s.

An English printing of the book published in America.

"Bacteriological Technique: A Guide for Medical Laboratory Technicians", by W. W. W. McEwen, A.I.M.L.T., F.R.M.S., with a foreword by Professor Sir Alexander Fleming, F.R.C.P., F.R.C.S., F.R.S.; 1949. London: J. and A. Churchill, Limited. Sydney: Angus and Robertson, Limited. 8" × 5½", pp. 300, with 70 illustrations. Price: 26s. 3d.

The purpose of the book is set out in its title.

"The Physiological Basis of Medical Practice: A Text in Applied Physiology", by Charles Herbert Best, C.B.E., M.A., M.D., D.Sc. (London), F.R.S., F.R.C.P. (Canada), and Norman Burke Taylor, V.D., M.D., F.R.S. (Canada), F.R.C.S. (Edinburgh), F.R.C.P. (Canada), M.R.C.S. (England), L.R.C.P. (London); Fifth Edition; 1950. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 10" × 7", pp. 1330, with 601 illustrations. Price: £5. 18s. 3d.

A completely revised and enlarged edition of a standard work.

"Parkinson's Disease", by Walter Buchler; 1950. London: Walter Buchler. 7" × 5", pp. 80. Price: 5s. 6d. (paper cover) and 8s. 6d. (cloth cover).

Written by a man with Parkinson's disease with the intention of helping his fellow sufferers.

"Handbook of Obstetrics and Diagnostic Gynecology", by Leo Doyle, M.S., M.D.; 1950. Palo Alto: University Medical Publishers. 7" × 4", pp. 254, with 45 illustrations. Price: $2.00.

A pocket manual for the practising doctor.

"Out of My Later Years", by Albert Einstein; 1950. New York: Philosophical Library. 8½" × 5½", pp. 292. Price: $6.00.

Contains chapters on philosophical, social, political and scientific subjects.

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE COMMONWEALTH SCIENTIFIC AND INDUSTRIAL RESEARCH ORGANIZATION.

THE Commonwealth Scientific and Industrial Research Organization, which came into existence on May 19, 1949, when the *Science and Industry Research Act*, 1949, was proclaimed, has issued its "first annual report" for the year ending June 30, 1949. At first this may appear to be something of an oddity. There is no oddity about it, however, because the new "organization" took the place of the Council for Scientific and Industrial Research. This in turn had superseded the Institute of Science and Industry in 1926. Now that the change has taken place and the organization has, as it were, got into its stride, it is well that medical practitioners, those on whom rests the care of the human beings, the men and women and children, of the Commonwealth, should be fully aware of the large army of research workers employed and of the great amount of money spent on science in relation to industry. Before the activities of the Commonwealth Scientific and Industrial Research Organization are mentioned it must be noted that the chief development that has occurred in the recent change is that there has been an alteration in administrative responsibility. The governing body of the organization is an executive of five persons and not a large council. We should imagine that this would make for easier working. The executive will have the assistance of an advisory council constituted in precisely the same way as the former Council for Scientific and Industrial Research. The present executive (at the time of the issuing of the report) consists of Dr. I. Clunies Ross (chairman), Dr. F. W. G. White (chief executive officer), Dr. S. H. Bastow, Mr. D. A. Mountjoy (part time), and Mr. H. J. Goodes (part time).

The powers and functions of the organization include "the initiation and carrying out of research in connexion with, or for the promotion of, primary and secondary industries in the Commonwealth or any Territory of the Commonwealth, or in connexion with any matter referred to the Organization by the Minister; the training of research workers; the making of grants in aid of pure scientific research; the testing and standardization of scientific apparatus and instruments, and the carrying out of scientific investigations connected with standardization; the collection and dissemination of information relating to scientific and technical matters; the publication of scientific and technical reports and periodicals; and acting as a means of liaison with other countries in matters of scientific research". For the purpose of carrying out its research work the organization has established a number of divisions and sections. The divisions include those on plant industry; entomology, animal health and production; biochemistry and general nutrition; soils; forest products; food preservation and transport; fisheries; metrology, physics and electrotechnology (comprising the National Standards Laboratory at Sydney); radiophysics; industrial chemistry; and tribophysics. These divisions have their headquarters in or near capital cities and branch laboratories and field stations at suitable places in every State. The sections deal with such subjects as irrigation, ore dressing, mineragraphic investigations, mathematical statistics, dairy research, building research, meteorological physics, coal research, wild life survey, and wool textile research. Regional centres have been established by cooperative research units with officers of the appropriate specialist divisions when problems of a particular district have had to be attacked. The reports on the work done in the several spheres are full of interest; unfortunately such a great deal of ground has to be covered that where the reader looks for detail he is likely to be disappointed. As will be seen from the divisions and sections which have been named, a good deal of investigation centres round what does not appear to be of immediate benefit to man's well-being—metrology, radiophysics, forest products, tribophysics, ore dressing, mathematical statistics and so on. Of course, a moment's reflection will discover an application for all these aspects of work. The average citizen, if there is such a person, will at once see the value of research in animal health and production, in biochemistry and general nutrition, in fisheries, in plant industry, in soils and in entomology. Most Australians will realize the importance of research in wool production and textiles and in the possibility of developing new methods of rabbit control. It is with this last-mentioned activity that the wild life survey is being undertaken. This survey has two objectives—first of all, to acquire detailed knowledge of rabbit ecology under the variety of conditions obtaining in the Continent; secondly, to obtain a better understanding of the value and limitations of standard control methods, and to determine whether the efficiency of any of them can be increased. A great deal of the activity of this organization is connected with the production and handling of animal and vegetable foods. In this regard it is important to note that steps are to be taken to further "the major agricultural development of the future" in northern Australia. Conferences have taken place between the Australian Meat Board, the Queensland Department of Agriculture and Stock and the Commonwealth Scientific and Industrial Research Organization, and plans for detailed work have been made, particularly on the improvement of the cattle pastures of Queensland and for the development and study of existing and new cattle breeds more suited to the north. This, we are reminded, is important not only to Australia from the economic aspect,

but also as a contribution to the solution of the world's major social problem, the provision of adequate food supplies.

Any person interested in the subject will naturally ask what all this activity costs. The expenditure for the year totalled £1,931,785. Of this amount £143,206 was contributed other than directly from the Commonwealth Treasury and this amount included £52,570 expended from the Wool Industry Fund. In addition the sum of £186,285 was expended on wool and textile research from funds derived as a result of the passing of the *Wool Use Promotion Act*, 1945. This means that the work of the organization (and of its predecessor) for one year was well over £2,000,000. It should finally be noted—and the point should be emphasized—that outside bodies have provided donations for cooperative research. These have included the Commonwealth Bank of Australia, the Australian Dairy Cattle Research Association, the New South Wales Department of Agriculture, the New South Wales Water Conservation and Irrigation Commission, the Cement and Concrete Manufacturers' Association, the Queensland Meat Industry Board, the National Gas Association, the Dried Fruits Control Board, the timber industry, and the pulp and paper industry.

The people of Australia, and the medical practitioners of the whole Commonwealth with them, have reason to be proud and appreciative of the work done for them in industrial science. This is something that everyone can comprehend; it is something the value of which should be impressed on every growing boy and girl when they are old enough to understand. If they can be made to appreciate what research means they will the more readily realize that knowledge about man and his diseases is incomplete. In this respect politicians should become as adolescents and they will learn that human health and happiness have to be "promoted" by the study of disease and its accompaniments; they will make the endowments of medical research at least comparable with those of science and industry. The report of the Commonwealth Scientific and Industrial Research Organization gives an excellent object lesson.

Current Comment.

ESSENTIAL HIRSUTISM.

EXCESSIVE growth of hair has been frequently reported as a feature of certain endocrine disorders of women, Cushing's syndrome being the best known. In other instances it can be described only as essential or idiopathic hirsutism. Little is really understood about hair growth, either normal or abnormal, but hirsutism in women is sufficiently distressing to warrant more intense investigation. A contribution to the subject of interest has been made by J. L. Callaway, J. T. Wortham, E. C. Hamblen and A. A. Salmon,[1] who studied a group of patients with essential hirsutism and contrasted them with patients suffering from known endocrine disorders to see if any significant endocrinological factors could be correlated with the hair growth. The hirsutism of those with the essential type was severe enough to require shaving or to cause psychic disturbances. The detailed findings cannot be reproduced here, but Callaway and his colleagues point out that they emphasize commonly held impressions, namely, that the intensity, amount and distribution of hirsutism are not diagnostic evidence of endocrinopathy; on the other hand patients with established virilizing syndromes may have unimpressive hirsutism. Thus patients with hirsutism warrant capable medical consideration, but complicated special endocrinological studies are not necessarily required. The differentiation of essential hirsutism from that associated with endocrine disease depends, in the view of these investigators, more on clinical than on laboratory investigation. If, they state, a woman with hirsutism has cyclic uterine bleeding of normal duration and has no symptoms or signs of virilization, it is unlikely that there is any endocrine cause for her hirsutism. If it can be proved, by basal temperature graphs or by endometrial biopsy, that this patient has normal ovulatory function, it becomes practically certain that no endocrine disease exists, since normal ovarian function is not compatible with the virilization syndromes. The symptom complex of gravest prognosis is amenorrhœa for twelve months or longer and hirsutism. A progressive decrease in frequency and amount of flow, occurring with hirsutism, warrants definitive studies. Other probably significant associated symptoms are obesity, coarsening of the voice, psychosexual changes and alterations in libido. Careful consideration of the symptoms and physical examination usually suffice. Special studies which may help include the recording of basal temperature curves and examination of endometrial biopsy specimens. Hormonal studies have little value in differentiation at present, the role of endocrine factors in "idiopathic" hirsutism remaining still obscure. By the same token, no specific endocrine or other treatment is known for essential hirsutism. The empirical use of œstrogens not only may fail to relieve the hirsutism, but also may bring about significant alteration in ovarian function, with resultant menstrual irregularities, as well as aggravation of undiagnosed local gynæcological disease, such as carcinoma or endometriosis. The only treatment suggested for essential hirsutism is either that the patient should forget and ignore the condition or that palliatives should be used, such as shaving, manual epilation, bleaching or electrolysis, or both. It is to be hoped that further investigations will show the way to an understanding of the cause of this unfortunate condition, with the corollary of rational and effective treatment. It is not unlikely that an endocrine factor is closely concerned.

[1] *Archives of Dermatology and Syphilology*, October, 1949.

INTRAGASTRIC DRIP THERAPY FOR PEPTIC ULCER.

IN 1932 A. Winkelstein devised and introduced intragastric drip therapy for peptic ulcer. It was based on two ideas: first, that whatever the ultimate cause of peptic ulcer might be, free hydrochloric acid *plus* pepsin was a prime factor in the development and persistence of the lesion; second, that methods which were most effective in the neutralization of the free acidity throughout the twenty-four hours of the day were most likely to be successful in the medical treatment of peptic ulcer. In 1942 Winkelstein, A. Cornell and F. Hollander[1] reported in summary the results of ten years' experience with the method and described it as "an excellent method for the adequate control of interdigestive acidity", which was easily learned and gave the patient a means of self-therapy at home for long periods during the night without interfering with his daily work. They put forward as reasonable requirements for any new form of therapy for peptic ulcer that it should be rational and practical and produce prompt and more persistent results than other forms of therapy. After their ten years' experience they believed that the drip therapy (with milk and sodium bicarbonate or alumina gels) satisfied those criteria.

Recently support for the method, with certain topical reinforcements, has been offered by A. M. Clark,[2] who

[1] *The Journal of the American Medical Association*, November 7, 1942.
[2] *The Lancet*, March 11, 1950.

points out that it requires less attention from the nursing staff than other methods and thus enables a small staff to care for more patients—an important point in these days of nursing shortage. The results of the method appear, in Clark's hands, to have been satisfactory. He lists as the indications for it active peptic ulceration, recent hæmatemesis or melena, and the need to build up a patient's general condition, as for a surgical operation. His technique is given in detail in his paper, which should be consulted at first hand by those anxious to try the method. Milk is regarded as the ideal solution for use, the aim being to give 100 ounces (citrated with 40 grains of sodium citrate to the pint) in twenty-four hours. However, difficulties of supply are considerable in England, and Clark used, as the best substitute, a solution of magnesium carbonate in a 1:3 dilution of the British Pharmacopœia solution delivered at the rate of 80 ounces per twenty-four hours; its disadvantages are that it has not the nutritive value of milk and is laxative, though the laxative action is overcome by the addition of 15 minims of tincture of opium to the drip reservoir twice daily. Except for very seriously ill patients, those receiving drip therapy have a routine "gastric" diet, which excludes gross roughage, chemical irritants and fried foods; the tube is removed for the meal period. Three weeks is the maximum period for which most patients can tolerate the treatment; they spend another week in bed and if symptom-free are then allowed up gradually. If relapse is anticipated the patient is advised to use the method at home at night and during week-ends. In Clark's series of 35 patients, the most notable feature was the almost instantaneous relief of abdominal pain. In 19 of 29 patients with radiological evidence of ulcer, that evidence disappeared in from one to sixteen weeks after the drip therapy had ended. A substantial proportion of the unsatisfactory results were associated with chronic ulcers. For the acute and subacute condition it appears to offer useful possibilities, though it has always to be borne in mind that peptic ulcer has elusive factors in its ætiology which create possible fallacies in the assessment of any method of treatment.

BENJAMIN BYNOE, NAVAL SURGEON.

THE name of Benjamin Bynoe, Esquire, M.D., Staff Surgeon, R.N., has been caught up in the charting of Australian geography and in the weaving of Australian history, and might well have appeared prominently in the naming and description of our fauna and flora but for certain circumstances. For all that, he never lived in Australia, though that, too, might have been, and today he is to most not even a name. Few see his name attached to a bay, an inlet and a river on the charts of Australia's northern coastline; few know that he visited Australia twice as surgeon on H.M.S. *Beagle* (once with Charles Darwin on board and once for a six-year survey of practically the whole Australian coast) and a number of times with convict ships, and that he contributed to J. L. Stokes's book "Discoveries in Australia"; fewer still have heard of *Acacia Bynoeana*, his only botanical memorial, or would know, if J. B. Cleland[1] had not told them, that Bynoe first captured the Gouldian finch and narrowly missed solving the problem of the birth of the kangaroo.

However, amends have been notably made by J. J. Keevil,[2] who has searched diligently for his facts, though he appears not to have had access to some of the Australian sources mentioned by Cleland. Bynoe, Keevil states, was born in Barbados in 1803, became a member of the Royal College of Surgeons of England in 1825, after obtaining his "London Diploma", and later in the same year was appointed an assistant surgeon in the Royal Navy. Four weeks after his appointment he was directed to join H.M.S. *Beagle*, which was to be his home for eighteen years. On his first voyage in the *Beagle*, which was accompanied by H.M.S. *Adventure*, a survey was made of the southern part of South America. Bynoe took an active part in this survey, during which his name was given to a cape and an island, and he gathered geological and other specimens, which are in the museums of the Geological Society and the Zoological Society, in London.

The next voyage of the *Beagle* (1831-1836) was to make her famous, for she carried Charles Darwin, as the guest of Captain Robert Fitzroy, who commanded her. Of that voyage, which Darwin described as the most important event in his life, Keevil provides an interesting account. It seems to have been important to Bynoe also, in the medical problems he encountered, in the part he played in surveys, and in his association with Darwin. The main point of local interest is that the *Beagle* called at an Australian port on her way home.

In 1837 the *Beagle* was sent on surveying service in Australian waters, a task which lasted till 1843. The continent was circumnavigated, and many parts, including much of the northern and north-western coast, were investigated in detail. Bynoe's name was attached to a bay on the west coast of the Northern Territory, and to an inlet and a river in the Gulf of Carpentaria. He carried out his medical and surgical work satisfactorily, earning a reputation for care and kindness, and also made a considerable collection of specimens, for which he received little credit.

Keevil suggests that when the *Beagle* paid off at Woolwich in October, 1843, Bynoe might have done as other young ship's surgeons had done and followed up his extensive shipboard research into natural history with a distinguished career ashore in this field. For some reason he did not, and others, who could have been expected to be interested, seem to have neglected his collection of specimens and to have done little justice to the collector. Bynoe was by now married, but the next year (1844) he was at sea again in the melancholy role of surgeon superintendent on the convict ship *Blundell*, bound for Norfolk Island. On the *Blundell* and on a second convict ship, the *Lord Auckland*, in 1846, Bynoe seems to have maintained his reputation for professional skill and personal kindness, but life both at sea and ashore was proving rather depressing. His wife accompanied him on the *Lord Auckland*, whether with the idea that they should settle in New South Wales or not is not clear, but Bynoe had to be put ashore with pneumonia at the Cape of Good Hope, and on his recovery they returned to England. In 1847 Bynoe was sent to Ireland to aid in the relief of the Irish who were in a desperate state from destitution, famine and epidemic disease. He did what he could, despite his own illness and a certain amount of administrative frustration, but by the end of the year was again on board ship. He worked on a series of ships, but it was an unhappy time, with the care of convicts, service in the yellow fever zone of South America and financial worries. After a period of naval service ashore, he was placed on the retired list in 1863 and died in 1865. His estate amounted to less than £450 and he was unknown, although J. L. Stokes had paid a tribute to him in his book "Discoveries in Australia" (published in 1846), acknowledging his contribution to the work in papers on the Australian aborigines, on the climate and on marsupials; Joseph Hooker had referred to him briefly, but in complimentary terms, in his book "Botany of the Antarctic Voyage". Meantime Bynoe's companion of the *Beagle*, Darwin, and two other naval surgeons, Hooker and Huxley, had attained scientific eminence. It is interesting to wonder by just how much Byrnoe missed something of the greatness of these others, and whether it was just diffidence or modesty, or the possibility that he was not interested in botany, that prevented him from working himself on his collection on the *Beagle* rather than prepare the specimens and hand them over to be neglected for so many years. He was, it would appear, a great man of simple qualities, and Keevil has done well to rescue his name from the geographical records and to tell us something of his work and personality.

[1] THE MEDICAL JOURNAL OF AUSTRALIA, April 29, 1950.
[2] *Journal of the Royal Naval Medical Service*, October, 1949.

Abstracts from Medical Literature.

PÆDIATRICS.

Typhoid Fever Treated with Chloramphenicol.

WILLIAM A. REILLY AND J. A. HARREL (*The Journal of Pediatrics*, April, 1950) report a series of 14 children with typhoid fever treated with chloramphenicol. The dosage given was an initial dose of 50 milligrammes per kilogram of bodyweight, followed by 125 milligrammes for small children and 250 milligrammes for larger children every two to three hours till fever disappeared (a period of two to four days), and then every four to six hours during four to six days of normal temperature. Two relapses occurred, but the patients responded quickly to further administration of the drug. There were no drug reactions, disease complications, sequelæ or known resultant carriers.

Resistant Rickets.

SMITH FREEMAN AND IRVEN DUNSKY (*American Journal of Diseases of Children*, March, 1950) discuss resistant rickets. They state that widespread use of vitamin *D* to prevent and cure rickets has brought to light a small group of patients who have the developmental features of rickets, but who fail to respond to ordinary doses of vitamin *D*. Vitamin *D* decreases the loss of calcium in the stools, probably by increasing absorption and perhaps by decreasing excretion. This also increases phosphorus absorption, for much of the calcium is in the form of calcium phosphate. It also acts on the renal tubules to increase tubular reabsorption of inorganic phosphorus. The rhachitic child usually has a normal serum calcium content and a low serum phosphorus content, the product of these two values in milligrammes *per centum* being less than 30. It has been shown that rhachitic bones will heal if exposed to a fluid containing the correct proportion of calcium and phosphorus. If vitamin *D* in large doses is given to animals on a diet of low calcium and phosphorus content, decalcification of bones will take place, to cause a rise in the amount of plasma calcium and phosphorus which is in turn excreted. The optimal effect of vitamin *D* is seen where the ratio of calcium to phosphorus in the diet is between 1·0 and 1·5, a ratio found in cow's milk. Resistant rickets that does not respond to ordinary doses of vitamin *D* tends to have a familial incidence, and tends to occur in childhood rather than in infancy or to persist to childhood. It does not seem to be due to faulty absorption of vitamin *D* from the bowel, for high blood levels of vitamin *D* are found. Extremely large doses of vitamin are required to promote healing (even up to 1,000,000 units daily), and relapse is likely to occur unless a large maintenance dose is given. While such treatment is being given, bone operation or prolonged immobilization in bed is dangerous, for osteoporosis, hypercalcæmia and calcium deposition in kidney with impairment of function may result. The underlying metabolic defect in resistant rickets is not clear. Some work indicates a failure of renal tubular reabsorption of phosphorus, some an increase in the rate of glomerular filtration of phosphorus. There is evidence of parathyreoid hyperplasia, but this is found, too, in ordinary rickets and in any condition tending to cause decrease in serum calcium content. Whatever the defect underlying the disease, there is usually a response to large doses of vitamin *D* given by mouth, and maintenance doses are required. The margin between the effective dose and a toxic dose that would result in hypercalcæmia, renal calcification and azotæmia may not be great. The disease sometimes ceases spontaneously when growth of long bones ceases, but sometimes the disease persists as osteomalacia in adult life. It must be distinguished from osteochondrodystrophy, eccentro-osteochondrodysplasia (Morquio's disease), renal rickets, *osteitis fibrosa cystica* and Fanconi's renal tubular rickets (in which the tubular defect is more severe with failure to reabsorb not only phosphorus but also sugar and amino acids). A case of resistant rickets with detailed metabolic studies is reported.

Gastro-Œsophageal Intussusception.

HARRY J. COHEN *et alii* (*The Journal of Pediatrics*, March, 1949) report a case of retrograde intussusception in a male infant, aged sixteen months, in whom the pyloric end of the stomach was invaginated into the œsophagus. The outstanding symptom was hæmatemesis with the picture of an unrelieved high intestinal obstruction. *Post mortem* no ætiological factors were found that could have caused such an intussusception.

Hyaluronidase.

JOSEPH SCHWARTZMAN AND MORRISON LERBARG (*The Journal of Pediatrics*, January, 1950) discuss the use of hyaluronidase as a "spreading" agent. They state that this substance is found in many bacteria, and also in leech extracts, bee, snake and spider venoms, and spermatozoa. It acts on and depolymerizes hyaluronic acid, which seems to hold water in tissue spaces, binds cells together in a jelly-like fashion and has been found in many body tissues. The toxicity of hyaluronidase is exceedingly low, no ill effects from its administration having been observed on temperature, pulse, blood pressure, blood count, urine or liver. With earlier products some allergic reactions resulted, and a positive result on skin test was given by 9·3% of patients tested. With later products this has been reduced to 0·08%. The greatest use of hyaluronidase has been in facilitating hypodermic administration of solutions of saline and glucose, in which in an average case 250 to 300 millilitres of electrolyte solution could be given within eighty minutes, and repeated frequently. Without hyaluronidase this would take some one hundred and eighty minutes, and the clysis could not be repeated within eight hours. The same area could be used for up to five days, but tended to become inflamed after the third day. The first amount injected was absorbed more rapidly than subsequent ones. Isotonic or hypotonic solutions were absorbed more rapidly than hypertonic ones. There is some evidence that in diseases with a plasma protein content of less than 5·5% the effectiveness of hyaluronidase is much reduced. The substance has also been used with some success in aiding the diffusion of local anæsthetics, penicillin, streptomycin and the dye used by intramuscular injection for pyelography, and for disintegrating plugs of wax in ears. There is some evidence that salicylates inhibit the action of hyaluronidase. It has been suggested that some of the phenomena of rheumatic fever are due to an increased hyaluronidase activity, and that salicylates give relief by their inhibiting action.

Chorio-Retinopathy Associated with Other Evidence of Cerebral Damage in Childhood.

A. B. SABIN AND H. A. FELDMAN (*The Journal of Pediatrics*, September, 1949) have shown that the quantitative in-vitro toxoplasma dye and complement-fixation tests give certainty to the diagnosis of congenital toxoplasmosis. They found that chorio-retinopathy, associated with other evidence of ocular and cerebral damage or defects and not due to toxoplasmosis, was not infrequently encountered in infancy and early childhood. When infantile chorio-retinopathy was associated with positive serological findings for toxoplasmosis, the incidence of grossly perceptible cerebral calcification in X-ray films of the skull was approximately 90%, while in a group of cases with chorio-retinopathy and negative serological findings for toxoplasmosis, the incidence of cerebral calcification was only 5%. The authors state that available data suggest that the syndrome of infantile retinopathy, without cerebral calcification or evidence of toxoplasmic infection, is due to defective development rather than to destructive necrotic lesions. They also draw attention to a syndrome characterized by extensive destruction of brain tissue, hydrocephalus, diffuse cerebral calcification and chorio-retinopathy associated with bizarre degenerative changes in small blood vessels and distinguishable from toxoplasmosis by serological tests. Present data indicate that normal subsequent children have been born in all instances in which the diagnosis of congenital toxoplasmosis could be made with certainty.

ORTHOPÆDIC SURGERY.

Slipped Capital Femoral Epiphysis.

A. KLEIN, R. J. JOPLIN, J. A. REIDY AND J. HANELIN (*The Journal of Bone and Joint Surgery*, January, 1949) present a study of 31 patients with slipped capital femoral epiphysis, four with bilateral involvement, representing a total of 35 hips nailed *in situ* because the amount of slipping had been less than one centimetre. After an average follow-up period of thirty-two months, the average index of motion was 90% of normal, and the average percentage of normal hip function was 96. The function of the hip was appraised by designating a factor for each motion according to its importance; the amount of demonstrable motion was multiplied by this factor to give a product for that particular motion, the sum of these products representing the "index of motion". Since such an index of motion does not include pain or limp, equal value was assigned to the index of motion, absence of pain and absence of limp, the average of these being called the percentage of true normal function of the hip. The authors state

that in this series, in which nailing was performed *in situ*, traumatic arthritis or aseptic necrosis of the femoral head had not occurred. Sixteen patients with slipping of more than one centimetre were treated with arthrotomy, osteotomy through the epiphyseal plate, replacement of the head to its anatomical position in relation to the neck, and lateral nailing for fixation. After an average follow-up period of thirty-three and a quarter months, the average index of motion was 85% of normal, and the average percentage of normal hip function was 92. The authors believe that replacement of the head to its normal valgus relationship with the neck can be effected only at the site of the original displacement, namely, the epiphyseal plate. They emphasize that two additional basic requirements are necessary to obtain the results shown: the hip should be entered through an incision across the capsule, over the anterior portion of the epiphyseal plate, thus sparing the *ligamentum teres* and the posterior, superior and inferior portions of the visceral capsule; the osteotomy is performed through the epiphyseal plate, which is avascular, without any of the neck being sacrificed. These details minimize the possibility of damage to the circulation of the head. In this series, traumatic arthritis has been encountered in only two cases. The authors have noted radiological evidence of fusion of the epiphysis within four to eight months after nailing in the open-reduction cases and within six to eighteen months in the cases of nailing *in situ*. In the latter group, the nailed epiphysis fused eight to twenty-one months sooner than that on the uninvolved side. With open reduction, the nailed epiphysis fused five to thirty-five months before that on the uninvolved side. Apparently, osteotomy through the epiphyseal plate accelerates fusion more rapidly than fixation by nailing *in situ*. In four cases of nailing *in situ*, growth continued away from the end of the nail.

Stabilizing Operations on the Foot.

R. L. PATTERSON, JUNIOR, F. F. PARRISH AND E. N. HATHAWAY (*The Journal of Bone and Joint Surgery*, January, 1950) have investigated results of 305 foot stabilization operations. They found that stabilization was successful in 82% of cases. Residual deformity or pseudarthrosis accounted for 51 of 55 failures. Over two-thirds of the residual deformities resulted from undercorrection at the time of operation. Listed in order of frequency, removal of the plaster before solid fusion had occurred, failure to align the foot with the ankle joint, pseudarthrosis, and loss of position in plaster or at the change of plaster, were much more common as the causes of recurrence of deformity than muscle imbalance. The results were more successful when three joints were resected. Mid-tarsal wedge osteotomy resulted in the highest percentage of failure and did not correct the foot varus. Stabilization carried out on children up to and including eight years of age produced 47% of failures. In the group from nine to eleven years, inclusive, the results were as good as those in the series as a whole. Varus and drop-foot were the most frequent deformities that remained undercorrected. Stabilization, with the exception of panarthrodesis, cannot be expected to correct drop-foot. Pseudarthrosis occurred in 18% of cases. Approximately one-fifth of the patients had such severe pain that a rating of failure was mandatory. Of the 56 pseudarthroses, 50 were at the talonavicular joint. Three-quarters of the revision procedures produced satisfactory results, indicating that repeated intervention was justified. The foot operated upon was on the average eleven-sixteenths of an inch shorter than the foot not operated upon. The same foot-length discrepancy was present in 80 patients who had been operated upon before the age of twelve years. The authors state that these operations had been employed for many conditions, but by far the most common, in order of frequency, were poliomyelitis, spastic paralysis, Friedreich's ataxia, congenital club-foot and painful flat-foot.

Vitallium-Cup Arthroplasty of the Hip Joint.

A. GIBSON (*The Journal of Bone and Joint Surgery*, October, 1949) has reviewed 111 cases of vitallium-cup arthroplasty of the hip joint. Of the patients 89 had osteoarthritis of the hip and all were suitable for this form of treatment. The results with atrophic arthritis proved less satisfactory, and with Marie-Strümpell disease were wholly disappointing. Of two cases of tuberculosis, in one the procedure was undertaken deliberately and in the other the infection was a belated discovery. In both the treatment failed; both patients had arthrodesis later with satisfactory outcome. Of three cases of ununited fracture of the neck of the femur, in one the result was satisfactory and in the other two it was not. Of two patients with slipped epiphysis, one, aged thirty-three years, was at the last report free from pain, but was still using a crutch. The other, aged sixty-one years, suffered dislocation of the cup from the acetabulum, the roof being very sloping; fusion of the joint was performed later. Of two cases of septic arthritis, gradual stiffening occurred in one and at a later date the head of the femur was excised; the other followed a severe war injury, and the result, while not spectacular, is regarded as a success. A good result was obtained in a case of fracture-dislocation. Of the 111 cases, the results were found to be good in 65, satisfactory in 16 and bad in 19; in 11 they were unclassified. In every case the hip joint was exposed by the posterior approach, based upon that described by Kocher. The author found that this approach was very simple, thorough, extensible to any degree, and almost bloodless; it was unaccompanied by shock.

Kienbock's Disease.

A. DORNAN (*The Journal of Bone and Joint Surgery*, November, 1949) has reviewed the results of treatment of 43 patients with Kienbock's disease under his care during the past ten years. The author did not find that relative shortening of the lower end of the ulna was present in a high percentage of cases. Of the 43 patients, 40 were male and three female; 38 were heavy workers, including 25 coal-face workers, and five were light workers, including one housewife. The right wrist was affected in 24 cases, the left in 19. Of 27 patients treated conservatively, 22 were treated by immobilization for an average period of three to four months, two by rest and physiotherapy, and three by no specific treatment except a period of rest from work. Sixteen patients were treated by excision of the lunate bone, six as a primary measure and ten after a period of conservative treatment had failed to give relief. Of the patients treated conservatively 63% returned to full work; the three receiving no specific treatment had good or excellent results; of the two treated by physiotherapy alone, one had a good and the other a fair result. Of patients treated by excision of the lunate bone, 69% returned to full work. One patient with a poor result was treated subsequently by arthrodesis of the wrist. It was thought that the group of patients under the age of forty years who were treated conservatively was on the whole comparable with the group in which excision was undertaken. In cases of successful treatment by removal of the lunate bone, a striking result was the frequency of relief from aching pain; the strength of grip, usually diminished just after the operation, gradually returned, though in some cases only after many months. In none of the cases regarded as satisfactory has the condition deteriorated. In the author's opinion removal of the lunate bone is justified in the few cases in which there is no gross osteoarthritis, and in which aching pain persists after efficient immobilization for three or four months.

Fractures of the Forearm in Adults.

R. A. KNIGHT AND G. D. PURVIS (*The Journal of Bone and Joint Surgery*, October, 1949) have studied the end results in 100 cases of fresh fractures of both bones of the forearm in adults and found a high incidence of unsatisfactory results. The results following manipulation were, in general, unsatisfactory; this applied in 71% of cases. It was found that solid union required a longer period of time than is generally thought necessary, averaging five months. The authors believe that improper rotational alignment is an important causative factor in a high percentage of poor results. They state that transverse fractures in the middle and lower thirds of both bones of the forearm may, in some instances, be satisfactorily reduced by closed manipulation, if reduction is performed with correct rotational alignment. Fractures of the upper third of the forearm, and oblique or comminuted fractures at any level in the forearm, are best treated by open reduction and rigid internal fixation of both bones. Simple transverse and oblique fractures of the shafts may be plated; but comminuted fractures of the shafts are most satisfactorily treated by primary bone-grafting. Intramedullary fixation is a very satisfactory way of maintaining the reduction and alignment of complicated fractures of the shafts of both bones of the forearm, particularly where there is major soft-tissue injury and where other methods of internal fixation, such as plating and primary grafting, are contraindicated. External plaster fixation must be maintained until union is clinically and radiographically complete, regardless of the type of internal fixation employed. The authors point out that angulation of the radius and ulna during the period of cast immobilization due to atrophy of forearm muscles can be prevented by suspending the plaster cast by a sling incorporated in the plaster just below the elbow.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on March 4, 1950, at Warrnambool, DR. ROBERT SOUTHBY, the President, in the chair. The afternoon meeting took the form of a series of clinical demonstrations by members of the medical staff at the Warrnambool Base Hospital. In the evening DR. R. S. HOOPER, of Melbourne, read a paper entitled "Head Injuries". Part of this report appeared in the issue of June 3, 1950.

Radiological Signs of Bronchiectasis.

DR. A. PARK demonstrated from several series of skiagrams some of the radiological signs of bronchiectasis. He said that bronchiectasis was of two types: congenital, which was often associated with other malformation of the bony thorax or lungs, and acquired, which was the sequel to atelectasis and infection. Bronchiectasis was bilateral in 50% of cases and restricted to one lobe in 25% of cases. The X-ray appearance in plain films was that of coarse mottling towards the lung bases and filling up of the cardio-phrenic angle. Basal striations were increased; as a rule they ceased at the level of the diaphragm and did not pass below or regularly diminish in size or in branching as vascular markings did. Cavities might be hard to find; they tended to lie along translucent streaks of larger bronchi. Occasionally small fluid levels were seen. Fibrosis and atelectasis often accompanied bronchiectasis. A collapsed left lower lobe might be hidden behind the heart shadow, and a lateral view would be necessary to localize it. In lower-lobe collapse compensatory emphysema might mask bronchiectasis and give normal translucency at the costo-phrenic angle, but the broncho-vascular markings were seen to originate from the upper hilum, and normal lower-lobe markings below the diaphragm were absent. Saccular bronchiectasis and large isolated bronchiectatic cavities containing secretion might be obvious, so might upper-lobe bronchiectasis in tuberculosis, but X-ray evidence at times was most indefinite. Honeycomb lung was found only in advanced cases. If atelectasis persisted the possibility of bronchiectasis should be suspected. Regional pneumonitis and atelectasis masked dilated bronchi in a plain film, and secretions excluded air. If suppuration developed, a homogenous shadow appeared in the surrounding lung, in which ragged cavities might show up an abscess.

Dr. Park then described the appearances produced with lipiodol. In cylindrical bronchiectasis, if the bronchi were completely filled, they were of even density; when partially filled they appeared as translucent streaks with denser walls. Bronchi seen end on appeared as spots or rings. Terminations did not show the usual fine branching, but a few club-shaped, irregular, fingerlike processes took their place, and finer bronchi did not fill. Saccular bronchiectasis produced rounded, circumscribed shadows. With the patient in the erect position lipiodol pools with fluid levels might be seen, or if the cavity was full of fluid lipiodol formed a ring shadow. Cavities varied in size. Sometimes bunches of small grape-like shadows occurred, grouped round smaller bronchioles. In varicose bronchiectasis tortuous bronchi were seen with small cavities along their course; the condition might be unilateral. Dr. Park said that lipiodol rarely failed to enter bronchiectatic cavities, congenital or acquired; it usually entered congenital cysts; it frequently failed to enter abscess cavities, cavernous neoplasms and tuberculous cavities, the bronchus being blocked by secretion, granulation *et cetera*; it never entered emphysematous blebs or bullæ.

In conclusion Dr. Park pointed out that bronchiectasis should be borne in mind with slowly resolving or recurrent bronchopneumonia, with atelectasis which was slow in clearing, with unduly prominent broncho-vascular markings at the lung bases and variation from the normal pattern, and as one cause of blood-stained sputum. In suspected cases, serial films, followed by bronchography, were necessary, preceded where possible by bronchial drainage.

DR. RUSSELL HOWARD congratulated Dr. Park and the staff of the hospital on the excellence of the radiology. From the practical aspect for the thoracic surgeon the types were those associated with local lung disease that was active when treatment had to be directed to the local activity; the presence of local lung disease which was demonstrably inactive; and bronchiectasis not associated with other local pulmonary disease. He thought that the figures supplied by Dr. Park were suggestive of an unduly optimistic attitude; 50% were stated to be unilateral and 25% unilobar; those proportions were more likely to be 75% and 15% respectively in an extensive series of case histories, as had been worked out for him in a review of some 80 cases at the Children's Hospital about 1940. He added that the unilateral unilobar cases were the ones especially suitable for surgical treatment, but he was prepared to remove the whole of one lung, both lower lobes and perhaps a lingula. He said that recently, in America, broncho-lobar segments had been extirpated; but it should be appreciated that the surgeon not infrequently found evidence beyond that shown in the skiagrams of damage in portions of the lungs believed from the clinical and radiological studies to be free of disease. He added that, of course, minor degrees of bronchiectasis might resolve if there was not a continuing local cause. It was desirable that bronchograms should be prepared periodically in the study of the progress of each individual case.

DR. H. BOYD GRAHAM asked Dr. Park to say how many sessions were required for systematic outlining of the state of the bronchial divisions of the lungs. There was a tendency for partial lipiodol investigation to be undertaken, which begged the question as to the state of the rest of the pulmonary tissue and respiratory passages; and absorption of retained radio-opaque material was slow and interfered with the interpretation of subsequent outlining of the hetero-lateral lung if undertaken too soon.

Dr. Park, in reply, said that of necessity he had had to abridge his remarks; in some of the early cases he had been able to demonstrate progressive resolution of the radiological signs, especially when atelectasis was the underlying basis. He went on to say that, at times, the patients were non-cooperative and sedatives were required; the bronchial catheter was used and it was difficult to get rid of secretions the presence of which interfered with filling; by the use of screening, he endeavoured to pick out isolated areas, as the expert bronchoscopist could place the catheter precisely where it was required to deliver the lipiodol for that purpose; it was certainly better not to attempt to do both sides at the one time.

Suspected Miliary Tuberculosis Treated with Streptomycin.

DR. T. B. PATRICK presented a lad, aged fifteen years, who had been employed as a farm hand. His illness had commenced with a "cold" six weeks prior to his admission to hospital in September, 1949. He was treated symptomatically, but later became dyspnœic and cyanotic, complaining of pain in the chest on breathing. His temperature was 102° F., and crepitations were audible at both lung bases. His condition settled down rapidly, but X-ray examination a few days later showed a condition very like miliary tuberculosis. On his admission to Warrnambool Hospital, examination revealed an emaciated, rather dopey boy with a temperature of 102° F. Except for a palpable spleen, no other abnormality was detected. Streptomycin was administered intramuscularly and intrathecally to a total of 51 grammes in ten weeks. Other treatment was symptomatic. The temperature and pulse rate were normal within twenty-four hours of admission. The blood sedimentation rate was within normal limits throughout. The X-ray appearances were normal within sixteen days. The result of the Mantoux test was negative throughout. The result of an agglutination test against *Brucella abortus* was negative for a titre of 1:20. His weight had increased by 21 pounds. Since the completion of streptomycin therapy in mid-November, the boy had remained clinically well, but his temperature chart had shown a fluctuation from 97° F. in the morning to 99° F. in the evening. It had been noticed for some weeks that he suffered from hyperhidrosis, especially of the palms; he stated that that had been only since the onset of his illness. At the present time there was an abnormality in his gait which Dr. Patrick found difficult to describe, but he thought it to be due to eighth nerve trouble.

DR. ERIC CLARKE discussed the problem of diagnosis. While admitting that the result of the Mantoux test at the time of admission to the hospital might have been negative if the boy had tuberculosis, Dr. Clarke thought that it should have become positive later. Also, the pulmonary "tubercles" had cleared rather speedily; in one case that he had followed up in a two-year-old child, it had taken five or six months for radiological demonstration of the disappearance of the tubercles; in another case a residual diffuse fibrosis was found at the autopsy examination. He went on to say that from the pathological structure of the tubercles it could not be expected that they would resolve so promptly as in the case under discussion. He drew attention to the fact that the temperature one day was 102° F. and by the next day was normal. He considered that apart from the skiagrams there were no positive

points to support the diagnosis of miliary tuberculosis or any other form of tuberculous disease; the patient might have had acute bronchiolitis, but should be removed from the "tuberculosis" category; the vestibular apparatus was already damaged.

Vaginal Tumour Complicating Pregnancy.

DR. IRVING BUZZARD presented a married woman, aged thirty-five years, who, when first examined in September, 1949, was expecting the birth of her fifth child on October 10, 1949. Her previous obstetrical history was perfectly normal except that with her last pregnancy, twenty-six months previously, she had had a venous thrombosis of her left leg. At the antenatal examination on September 2, 1949, the fœtus was found to be lying in the transverse position, and this was turned into a vertex position. On September 9 antenatal examination again disclosed the fœtus in the transverse position, and it was again turned into a vertex position. On September 16 the fœtus was once again in the transverse position. In order to determine any fœtal or pelvic abnormality to account for the difficulty in maintaining polarization of the fœtus, X-ray examination was made. The radiologist made the following report: "Shoulder presentation—vertex on maternal right side. No fœtal abnormality seen. One fœtus of average size. Android-gynæcoid pelvis of ample proportions." Dr. Buzzard mentioned at this point that the patient lived 18 miles away and could visit hospital only once a week. He went on to say that on September 23 the fœtus was once again in the transverse position, and a pelvic examination revealed a large vaginal mass preventing entrance of the examining fingers to any distance. The mass was semi-solid and estimated to be about six inches in diameter. It was in the left lateral wall of the vagina, and the vaginal mucosa moved smoothly over it. A rectal examination revealed that the tumour was bulging into the rectum, and the rectal mucosa also moved smoothly. There had been no difficulty in defæcation. She was admitted to hospital and examined under general anæsthesia on September 27. Nothing further was learned; the cervix could be neither seen nor felt, and the tumour could not be felt above the pelvic brim. Vaginal delivery was impossible, so Cæsarean section was performed under local anæsthesia on September 28. Incision was made for the lower segment operation, and when the bladder was pushed away from the lower segment a mass of large tortuous veins was discovered. They were about the size of an ordinary pencil. Discretion was considered the better part of valour, and instead of the usual transverse incision, a longitudinal incision was made for delivery, extending from the lower segment to the body of the uterus. The child having been first polarized, delivery by the head of a living baby presented no difficulties. Before the transverse incision of the peritoneum over the uterus was closed, an attempt was made to see if the vaginal tumour was connected with the uterus. A tumour could be felt along the left side of the cervix for about one and a half inches. A groove could be felt, but whether or not the tumour was part of the cervix could not be determined, as the many veins limited the scope of the examination. The wound was closed in the usual manner, and the question of drainage of the lochia presented difficulties, as nothing was coming away *per vaginam*. The vulva was prepared, and a sterile sponge holder was inserted and opened between the tumour and the right vaginal wall. Lochia came away freely. For continuous drainage, a Magill's pharyngeal tube was inserted up the vagina and strapped to the thigh. The tube was changed twice daily. Positioning of the patient made no difference to the drainage, and she was sitting out of bed on the second day. As prophylaxis she was given 600,000 units of penicillin in doses of 50,000 units six-hourly. A biopsy of the tumour was performed on the sixth day and the pathologist reported that the material consisted of dense fibrous non-neoplastic tissue, containing, here and there, dilated blood vessels. Convalescence was uneventful, and involution was good. Subsequent examinations showed the tumour retrogressing in size, and on January 4, 1950, the uterus could be felt as distinct from the tumour. A speculum could be inserted and the cervix identified. It was decided to remove the tumour on February 7. Vaginal douches were given three-hourly for forty-eight hours, and 100,000 units of penicillin were injected before operation. With the patient in the lithotomy position, an elliptical incision was made over the tumour through the vaginal wall. It was noted that the wall was particularly thick. The capsule of the tumour was easily discernible, and the tumour was freed by blunt dissection, particular care being taken round the rectal area. It was eventually delivered with the aid of a volsellum. Surprisingly little bleeding occurred. A gauze drain was placed in the vagina for twenty-four hours, the bowels were confined for six days, and a further 500,000 units of penicillin were given. Sedatives were unnecessary. On the seventh day the patient complained of a little soreness after defæcation, and examination disclosed a hæmatoma in the old tumour cavity. This was evacuated on the following day. From then on convalescence was uneventful. The husband subsequently told Dr. Buzzard that coitus had taken place approximately five months before the birth of the child, and no difficulty had been experienced. Coitus would have been impossible post-natally. The following pathological report was made on the tumour: "Macroscopic. Solid tumour 9 cms. in diameter. A thin rim of outer wall encapsulating a softer centre in which there were some yellow patches and a few cystic areas. Microscopic. Sections through the tumour show that it is a fibromyoma in which fairly recent necrosis had been superimposed on old hyaline and cystic degeneration. There is no evidence of malignancy."

Dr. Buzzard explained that the patient had been presented because benign tumours of the vagina were infrequent, and as complications of pregnancy they were very rare.

DR. S. G. FITZPATRICK said that he had seen the pedunculated variety of tumour to which Dr. Buzzard had referred; it was a huge fibroid tumour protruding from the cervix before full term and it had presented as a foul necrotic mass; after successful Cæsarean section the mass was removed at a later operation, and it proved to be a fibroid tumour of the vaginal wall.

DR. MICHAEL KELLY mentioned that he was reminded of a case in which the "tumour" was a turnip, which was removed by the surgeon.

A MEETING of the New South Wales Branch of the British Medical Association was held on March 23, 1950, at the Rachel Forster Hospital for Women and Children, Redfern, New South Wales. The meeting took the form of a series of clinical demonstrations by members of the honorary medical and surgical staff of the hospital.

Diaphragmatic Hernia in a Child.

DR. B. T. EDYE showed a male infant, aged one year, who had been admitted to the hospital on July 30, 1949, with a history of vomiting at least one feed daily and a dry cough, often ending with vomiting, for five weeks. He was also listless, had screaming fits and was constipated. On physical examination of the child, the apex beat was found in the left intercostal space, half an inch to the left of the mid-line, and the heart sounds were on the right side. There were diminished percussion notes and breath sounds over the lower two-thirds of the left side of the chest. Occasional borborygmi were heard. X-ray examination after a barium meal on August 11 showed all the small intestine and most of the large bowel to be in the left side of the thorax. The stomach was very low and appeared to be rotated. Screening examination was not done. Because of the low position of the stomach it was thought advisable to attempt screening while the patient was drinking, to outline the œsophagus. On January 5, 1950, left phrenic crush and repair of diaphragmatic hernia were performed with anæsthesia of ether, cyclopropane, nitrous oxide, oxygen, curare and "Pentothal". The findings were a large pleuro-peritoneal hiatus on the left side, the contents of the left side of the chest consisting of the small intestine and colon to the splenic flexure. The patient's condition after the operation was satisfactory and he was discharged from hospital on February 3, 1950.

Neurofibroma of Chest.

Dr. Edye's second patient, a married woman, aged twenty-two years, had been admitted to hospital on February 28, 1950, with a history of frequent colds for two years and breathlessness on exertion. Physical examination of the patient revealed scoliosis, greater expansion on the right side of the chest than on the left, and diminished percussion note, vocal resonance and breath sounds on the left side over the seventh, eighth and ninth intercostal spaces posteriorly. An X-ray examination of the chest showed a well-marked opacity in the posterior portion of the left side, extending laterally from the bodies of the seventh, eighth and ninth vertebræ. The Casoni reaction was absent. On March 2, under "Pentothal", curare and gas and oxygen anæsthesia, excision of a tumour from the left costo-vertebral gutter was performed, involving removal of the eighth rib. The tumour was hard, roughly spherical, and measuring eight centimetres by eight centimetres by seven centimetres, and arose from the sympathetic chain, attached by a pedicle over an area of 3·5 by 3·5 centimetres.

The pathology report revealed that it was a neurofibroma. Post-operatively the patient developed a pleural effusion, which was drained, and a satisfactory recovery was made.

Icterus and Ascites in a Young Woman.

DR. WILLA NELSON showed a married woman, aged twenty years, who had a history of yellow eyeballs for nine months, swelling of the feet for eight months and swelling of the abdomen for one month. She had felt well throughout. At the beginning she noticed pale fæces for a short time. Her urine was dark. The main findings from physical examination were mild jaundice, gross ascites and moderate splenomegaly. The fæces appeared normal; the urine was dark and contained no bile. The liver could not be felt. There was no family history of jaundice. The results of investigations were as follows. The serum bilirubin content was five milligrammes *per centum*. The icterus index was 54. The Van den Bergh test yielded a direct positive and an indirect positive reaction. The result of a urinary urobilinogen test was positive to a dilution of 1 in 50. The total serum protein was 7·05 grammes *per centum*. The serum albumin content was 2·80 grammes *per centum*. The serum globulin content was 4·25 grammes *per centum*. The albumin-globulin ratio was 0·66:1·0. The serum alkaline phosphatase content was 39·6 units. The result of the thymol turbidity test was "two plusses". The plasma prothrombin time was 32 seconds. The prothrombin index was 78%. A blood count revealed 3,890,000 red blood cells per cubic millimetre, with a hæmoglobin value of 12·6 grammes (90%) and a colour index of 1·01, and 5950 white cells per cubic millimetre, 53% being neutrophile cells and 47% lymphocytes. The fragility of erythrocytes was normal. Examination of the ascitic fluid showed no evidence of inflammatory reaction; the protein content was 400 milligrammes *per centum*; the specific gravity was 1·008; the deposit contained at most one epithelial cell and two to five leucocytes per high-power field; no bile was found; no growth appeared on culture, and no acid-fast bacilli were found.

Dr. Nelson said that the provisional diagnosis, supported by the history, physical findings and investigations, was chronic hepatitis, following a clinically mild attack of acute infective hepatitis nine months previously. The picture at the time of the meeting was that of portal cirrhosis with ascites and secondary splenomegaly. The patient had received no treatment for her "jaundice" in the early stage and had continued her work and her usual activities until the ascites appeared. It was interesting to speculate whether rest during the supposed original attack of acute infective hepatitis, together with dietetic or other treatment, would have prevented the disease from passing into the chronic stage. The question of laparotomy and possible surgical treatment was discussed.

Neurosyphilis.

DR. EDITH ANDERSON presented two patients with neurosyphilis. The first patient, a married woman, aged sixty-four years, had been under investigation for three months for weakness, very severe pain in the back of the head in the early morning which passed off a few hours after rising, itchiness of the back of the right shoulder and no feeling in it when she scratched it, and aching and loss of power in the right arm. Her husband died of "valvular heart trouble" at the age of forty-two years. He had not had heart trouble for long. She had three children, aged forty-four years, forty-one years and thirty-nine years respectively, and had had no miscarriages. She was a fairly well-nourished woman, looking the stated age, with no rombergism and of normal gait. Physical examination revealed marked loss of power in the right hand, exaggerated knee jerks, absent ankle jerks and abdominal reflexes and a plantar response difficult to elicit but flexor. Her pupils were unequal, but did react to light and accommodation. The circulatory system was apparently normal according to stethoscopic examination; the blood pressure was 130 millimetres of mercury (systolic) and 70 millimetres (diastolic). On the right side of the thorax posteriorly, there was an area of complete loss of touch and temperature sensation, extending from below the scapula to the mid-axillary line. There was some anæsthesia of the left side also, but it was much less. Blood examinations on December 15, 1949, and on February 9, 1950, revealed a weakly positive reaction to the Wassermann test and a positive reaction to the Kahn test. The cerebro-spinal fluid on March 3, 1950, yielded a weakly positive reaction to the Wassermann test and a content of 20 milligrammes of protein per 100 millilitres, and the colloidal gold curve was represented by the figures 5555432100-0. Dr. Anderson said that the patient's headache had improved after three injections of bismuth.

Dr. Anderson's second patient, a married woman, aged forty-three years, had reported to the out-patient department in 1947 complaining of heavy pressure in the epigastrium, vomiting attacks for years with headaches, tenderness of legs so severe that she could not bear her clothes to touch them, pains in the lower part of the body varying from place to place, and momentary unsteadiness of the feet causing falls. She had been to many doctors for three years without improvement. None of them had carried out a blood test. Ten years before she had had an ulcer on the leg which was treated as eczema. Her first husband had died after five years of marriage, twenty years previously. She had married again soon after; the second husband had always been well and his blood appeared normal. She was a plump, well-nourished woman, who did not look ill at first sight. On examination she had definite impairment of sensation to touch and heat along both legs, particularly on the outer side of the left thigh. Her eyes reacted normally, her ankle jerks were diminished, her abdominal reflexes were diminished, and her knee jerks were present. There was a great difference in the plantar reflexes, no reaction being elicited on the left side. The circulatory system and mental state seemed normal. Her blood tests yielded the following results: on April 20, 1947, an inconclusive Wassermann and an inconclusive Kahn reaction; on May 30, an incompletely positive Wassermann and a weakly positive Kahn reaction; on August 5, a positive Wassermann and a positive Kahn reaction. Her cerebro-spinal fluid yielded a positive Wassermann reaction; the colloidal gold curve was represented by the figures 5555543100, and it contained 50 milligrammes of protein per millilitre. After two injections of bismuth the lightning pains were relieved.

Congenital Syphilis.

Dr. Anderson then showed two patients with congenital syphilis, one being a child with congenital syphilis of the third generation, whose case was reported in THE MEDICAL JOURNAL OF AUSTRALIA on January 7, 1950. That child was now eleven months old, weighed eighteen pounds nine ounces, appeared healthy, and had made normal progress. The blood on January 16, 1950, had failed to yield the Wassermann and Kahn reactions. The Wassermann titre had gradually reduced in strength, but a positive reaction had still been yielded up to September, 1949. Dr. Anderson said that 1,700,000 units of penicillin were given in May, 1949, when the child was three months old. Infants' "Acetylarsen" was given weekly, and would be continued for another year. The mother had shown very little improvement in her blood titre after a year's treatment with "Acetylarsen" and bismuth. Penicillin would probably be tried soon. Her cerebro-spinal fluid was normal.

The second baby with congenital syphilis, a girl, had been taken to the doctor at the age of seventeen weeks, because the mother had noticed pallor, thrush in the baby's mouth, rash and blood-stained napkins. She had gained weight and then lost twelve ounces in the last week. On examination at hospital on February 28, 1950, the child was very pale, the urine test showed the presence of albumin and the blood yielded a positive Wassermann reaction. There was a little thrush in the mouth, a faint, dull red macular rash on the neck and inner surface of the arms and knees, and a palpable liver and spleen. X-ray examination of the chest and humeri showed a widening of the mediastinal surface, probably due to thymic enlargement, osteitis in both upper humeral shafts and periosteal thickening of the lower ends. Examination of the blood showed the presence of anæmia, the total erythrocyte count being 3,000,000 per cubic millimetre and the hæmoglobin value 65%. The urine contained red blood cells and pus cells and some albumin. Penicillin therapy was instituted, 15,000 units being given three-hourly up to a total of 1,285,000 units. It was stopped on the twelfth day because of the development of a pustular rash. Treatment with infants' "Acetylarsen" was started on March 4; it was given every four days, the average dose being 0·7 millilitre. The mother's blood yielded a positive Wassermann reaction with a titre of 20. She had no signs and no history of syphilis. The patient was the second child. The first had died at the age of three weeks in 1948, post-mortem examination showing that she had a grossly enlarged liver and spleen; the post-mortem diagnosis was "probably some blood dyscrasia". The mother's blood had not been tested before.

Apparent Herxheimer Reaction after Penicillin Therapy.

Dr. Anderson's last patient was a married woman, aged forty-one years, who had attended hospital with a pinkish, non-irritant, generalized rash. She was poorly nourished

and had had injections of "Anahæmin" for anæmia. She was a heavy smoker. She had had treatment for venereal disease twenty-one years before, with injections in the arms and buttocks for weeks. She had had extramarital contact five to six weeks before attending hospital, the partner saying he was healthy, but she did not believe him. Her knee jerks were normal, her eyes had a sluggish reaction to light and accommodation, and the circulatory system was apparently normal from stethoscopic examination. The blood on October 8 and November 1, 1949, yielded positive Wassermann and Kahn reactions and a blood count revealed 8,000,000 erythrocytes per cubic millimetre, a hæmoglobin value of 79%, a colour index of 0·97 and a leucocyte count of 6000 per cubic millimetre. The patient was given iron and started on treatment with half a millilitre of penicillin-in-oil for two days. The rash decreased, and she was then given one millilitre of penicillin-in-oil daily for eleven days, a total of 5,000,000 units of penicillin, until November 25. Bismuth was also given with the penicillin and was continued for seven weeks once weekly after the penicillin had been discontinued, that is, until January 12, 1950. She did not look very well, but was much better than when she was first examined, and there was much improvement in relation to the rash. On January 24 she was admitted to another hospital with a cerebral hæmorrhage and died.

Dr. Anderson said that the question to be considered was whether the penicillin produced a Herxheimer reaction and whether it would have been better to institute the usual custom in therapy of bismuth and iodides for an old case of syphilis. It was difficult to say whether the condition was a reinfection or not, but it appeared to be so. A somewhat similar death had been quoted in an American journal recently.

Correspondence.

"AVIATION MEDICINE: ITS THEORY AND APPLICATION."

SIR: In a recent number of your journal my publication on aviation medicine was reviewed. In this review surprise was expressed at the figure of 40% which was given as the incidence of air sickness on the occasion of second flights—and a figure nearer 4% was quoted as being closer to Australian experience.

I should be glad if you would permit me the courtesy of your columns to state that the figure of 40% was an unfortunate typographical error to which my attention has been repeatedly drawn, and which will be corrected in future editions. It should, as your reviewer correctly points out, have been 4% and not 40%.

With apologies for any trouble which may have been caused thereby.

Yours, etc.,

K. G. BERGIN.

31 Oakfield Road,
Clifton,
Bristol, 8,
England.
May 18, 1950.

FINAL RESULTS OF LONG-TERM THERAPY WITH METHYL THIOURACIL IN THYREOTOXICOSIS.

SIR: In reply to Dr. Francis Rundle's letter (THE MEDICAL JOURNAL OF AUSTRALIA, May 27, 1950), I would like to correct some of his statements, as apparently he has not followed the development of the work carried out by me with the "thio" compounds and especially with methyl thiouracil in thyreotoxicosis over the last seven years. There is no guess work about any statements I have published, as I have given full details and can substantiate my findings and conclusions.

As regards the maintenance of a *minus* metabolism, I am in good company, for E. B. Astwood is quoted by Paul Starr, of the University of Southern California, as claiming 85% of remissions, but "a state of hypothyroidism must be maintained for some months in order to obtain this result". In a personal communication to me, E. B. Astwood writes (April 2, 1947): "I was delighted with your statement that hypothyroidism must be induced and maintained in order that a remission be achieved. This has been our impression for some time" *et cetera*. Also in a letter from George Crile, junior (April 29, 1947), he states: "I am happy that your observations have borne out my theoretical conception of the interruption of the vicious circle and I hope that further experience will prove this is a correct conception." Apparently he is now quite convinced that it has been proven, according to his latest articles.

Also J. H. Means writes (April 15, 1947): "I think I may say that I find myself in substantial agreement with all you have to say about the thio drugs."

Coming to England, Professor H. T. Himsworth writes (June 4, 1947): "I may say at once that I think your views are probably correct. We have been groping towards a method of treating thyrotoxicosis entirely with thiouracil. In the light of our accumulating knowledge it seems to me very probable now that your scheme of treatment may be the better and I am sure you will not object to our following your advice and trying out this method." According to his article in the *British Medical Journal*, July 10, 1948, Himsworth did not carry out the method in full, but has used the smaller dosages mentioned by Dr. Rundle, and states: "Disappearance of the goitre has as yet been observed in less than 10% of cases and then only after two or more years of treatment"; yet with the dosage recommended by me this result is obtained in 64% of cases after eight to twelve months' treatment. Himsworth also states: "With few exceptions, all cases should first be given a full course of medical treatment and subsequent therapy determined by the results of this."

McCullagh and Sirridge, of Cleveland, used 200 to 300 milligrammes daily of methyl thiouracil in 100 cases, but state in future they intend using larger doses, which they will not lower for the maintenance period of treatment "as the maintenance of some degree of hypothyroidism during treatment appears to increase the chances of a lasting remission" (*Journal of Clinical Endocrinology*, Volume VIII, December, 1948). They give up to two grains of desiccated thyreoid daily to control any obvious hypothyreoidism.

Dr. Rundle refers to pressure symptoms being induced with "thio" therapy and quotes the peculiar case of a man who apparently developed myxœdema and abductor palsy of one cord after six weeks' treatment on methyl thiouracil, 150 milligrammes daily. Is one to infer that the abductor palsy was due to pressure? I have used "thio" compounds in over 1500 cases, but have never had any pressure symptoms other than local discomfort from an already enlarged gland, for which the exhibition of *Thyroideum Siccum* is indicated in any case.

As regards Dr. Rundle's reference to a rule of thumb, "operate on all nodular toxic goitres, treat the diffuse ones with thiouracil", this is the considered advice given by all the leading authorities in the United States of America, except the Lahey Clinic, who still say all cases require operation once hyperthyreoidism is under control. My cases are "high selected" only in that they are private or intermediate patients, and in 98% of them control of their thyreotoxicosis is obtained so readily that they are able to adjust their social and economic factors and even their psychoses without the necessity for admission to hospital with its expenses and loss of time and pay. Surely the 85% chance of full physical recovery is worth while trying without the risk of post-operative morbidity. Irrespective of social or economic status, most patients wish to get well with the least inconvenience.

As regards the mortality figures, my original remark was, "it takes years of experience to achieve a mortality rate of under 1%"—my own mortality rate in recent years is 0·1% and conforms to that of the average overseas experienced surgeon. In the five-year period 1943-1947, the overall mortality rate for surgically treated thyreotoxicosis at the Royal Prince Alfred Hospital was 3·3%, as pointed out in "A Fifty Years' Survey of Thyreoid Disease" by E. W. Gibson and myself, published in THE MEDICAL JOURNAL OF AUSTRALIA, July 30, 1949.

Prior to the advent of the "thio" drugs, the average mortality rate varied from 1% to 5% even in large clinics, but now it is down to the figures quoted by Dr. Rundle or even less, as in 1948 Bartels (a physician), of the Lahey Clinic, in "Appraisal of the Goitrogens" (*Transactions of the American Goitre Association*, 1948), recorded only one death in 1240 consecutive operations.

Thanks to Astwood, as well as to modern anæsthesia, the young surgeon of today knows little if any of the trials, tribulations, terror, physical strain and mental stress that older surgeons experienced in developing the surgery of goitre.

J. H. Means, in a recent article, "Clues to the Ætiology of Graves's Disease" (*The Lancet*, September 24, 1949), states: "Unless, perchance, the sensitivity of thyroid cells as end-organs is altered, which is possible but not proven, we may assume that the thyroid abnormality in Graves's

disease lies in the control of the thyroid gland rather than in the gland itself." "Thio" compounds act in the gland by reducing thyroxine production only, and it is this point I have tried to impress on those who treat thyreotoxicosis, which is a composite disease and not one only of the thyreoid gland; hence the necessity for adjuvant therapy and some attempt to control the pituitary-thyreoid axis as enunciated by Salter in 1940. It is not only the use of "thio" compounds, whatever their dosage, that compels attention. We are beginning to see daylight in the correct usage of "thio" compounds in this ailment, and there will be further advances of great interest in the near future; but do not let us get away from primary principles and honesty in the appraisal of results.

Yours, etc.,

225 Macquarie Street, Sydney,
May 30, 1950.

HUGH R. G. POATE.

MEDICAL HISTORY—DO WE USE IT WISELY?

SIR: I should like to comment briefly on one aspect of Dr. R. H. O'Brien's Jackson Lecture in the journal of May 20, 1950.

In this comparatively isolated and homogeneous population of about 20,000 people, I am sure that "leuchæmia" has increased in the last twenty years. I cannot give the diagnostic details that Dr. O'Brien so rightly insists upon, but from my intimate knowledge of the people and the practitioners, I can make due allowance for some of the possible fallacies. The lymphatic type is commoner than the myeloid, and instances of the former have recurred at all ages from infancy to well past middle age.

Yours, etc.,

E. A. WOODWARD, M.B., Ch.M., M.R.A.C.P.

Grafton,
New South Wales,
May 22, 1950.

PREGNANCY TESTS.

SIR: With regard to Dr. Poynton's letter in the issue of May 20, may I point out that I never said that the Xenopus test is impracticable in Australia. After all, it was the work of our laboratories that proved that this test could be successfully carried out in this country. All I did say was that large-scale breeding of the animals is impracticable in hospital laboratories, which is something quite different. The difficulties in obtaining the animals might not be as formidable now as they were some years ago, but Dr. Poynton should have heard what the head of the Melbourne firm, who established the commercial connexions with South Africa at our request, had to say about them. We abandoned the Xenopus test because we have now in the male Queensland toad test animals which are easily obtained at a fraction of the cost of Xenopus and need, as long as they can sit on a moist rag, no care whatever. A detailed report of this test will be published in an early issue of the journal.

With regard to Dr. Hagen's comments in the same number of the journal, I feel that they missed the point I was trying to make. First of all, I never spoke of frequently unexplained positive results; I did speak of rare, still unexplainable cases. Secondly, where Dr. Hagen professes to disagree with me he just emphasizes my contention. I stated that the variety of conditions listed in some text-books as causes of false-positive test results are surely not the direct causes of the results. This is just what Dr. Hagen elaborates at some length when he suggests that a tubo-ovarian abscess might destroy the ovaries to such an extent as to produce the menopausal or castration syndrome. I must confess that I have never seen such a case, but I would not say that it might not occur on some rare occasion.

Thirdly, however, I was not concerned at all with the menopausal or castration syndrome. It is true that mistakes in such cases can occur in laboratories which are not sufficiently aware of the different types of reaction elicited by the follicle stimulating factor ("FSH") and the luteinizing factor ("LH") of the gonadotrophic hormones respectively, but such laboratories should not perform pregnancy tests. Zondek and Aschheim, in their early publications on the subject, have already shown how these mistakes can be avoided; the female Xenopus has no monopoly as a reliable help in this direction.

There seems to be sufficient evidence that ovulation (and probably release of spermatozoa as well) will occur in the usual range of experimental animals only as a response to "LH", which, in quantities sufficient to induce ovulation in a test animal, is usually, but not invariably, produced by the placenta. On the other hand, "FSH", which is liberated in increased amounts by the pituitary in cases of ovarian failure, will on its own not bring about ovulation under the test conditions. On this basis false-positive results of this kind can be avoided.

I was, however, not really concerned with this type of case; I had in mind the rare occurrences in which one obtains a proper positive test result in the absence of pregnancy or any of the other well-known conditions that produce, *per se*, increased amounts of "LH". These cases are quite rare and usually defy an easy explanation, and certainly one on such well-known lines as Dr. Hagen suggested.

Yours, etc.,

H. F. BETTINGER.

Department of Pathology,
The Women's Hospital,
Melbourne.
May 24, 1950.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 29, of May 18, 1950.

AUSTRALIAN MILITARY FORCES.

Citizen Military Forces.

Northern Command: First Military District.

Royal Australian Army Medical Corps (Medical).—The resignation of 1/39012 Captain (provisionally) J. G. Watson of his commission is accepted, 16th March, 1950.

1/21022 Captain (Temporary Major) H. Glynn-Connolly, M.C., relinquishes the temporary rank of Major and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (1st Military District), 15th March, 1950. 1/39022 Captain (provisionally) P. J. Landy is seconded whilst undergoing post-graduate studies in the United Kingdom, 1st March, 1950.

Eastern Command: Second Military District.

Royal Australian Army Medical Corps (Medical): To be Captain (provisionally), 22nd March, 1950.—2/56833 Bruce Stevenson Hartnett.

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical).—3/50084 Major (Honorary Lieutenant-Colonel) H. McLorinan is appointed from the Reserve of Officers and promoted to the temporary rank of Lieutenant-Colonel, 2nd March, 1950. 3/51003 Major P. S. Woodruff is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (3rd Military District), 2nd November, 1948.

Western Command: Fifth Military District.

Royal Australian Army Medical Corps (Medical): To be Captain (provisionally), 30th March, 1950.—5/26429 Keith Wallington Hills Harris.

Tasmanian Command: Sixth Military District.

Royal Australian Army Medical Corps (Medical): To be Captains (provisionally).—6/9029 George Bertram Watkins, 16th March, 1950, and 6/15537 William Lindsay Elrick, 21st March, 1950.

The resignation of 6/15316 Captain (provisionally) D. E. Anderson of his commission is accepted, 31st August, 1949.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

1st Military District: To be Honorary Captain, 17th March, 1950.—Jeffrey George Watson.

1st Military District: To be Honorary Captains, 30th March, 1950.—Remo Cantamessa and James Geoffrey Toakley.

2nd Military District: To be Honorary Captain, 1st September, 1949.—Donald Edmund Anderson.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force: Medical Branch.

The resignation of Flying Officer G. McLean (409563) is accepted, 3rd May, 1950.

Citizen Air Force: Medical Branch.

The appointments of the following officers are terminated on demobilization: Temporary Squadron Leader (Acting Wing Commander) N. M. Kater, M.C. (267549), 4th April, 1950, Temporary Squadron Leader P. H. Cowan (257700), 14th April, 1950, Flight Lieutenant P. J. White (257696), 12th April, 1950.

Reserve: Medical Branch.

The appointment of Flight Lieutenant R. M. O'Donohue (267672) is terminated, 14th November, 1948.

Medical Prizes.

THE STAWELL PRIZE.

THE Stawell Prize, a memorial to Sir Richard Stawell, is open for competition. The amount of the prize is £30.

The conditions are as follows.

1. The prize shall be awarded to the writer of the essay adjudged to be the best on a subject selected annually.
2. The subject for 1950 is "Rheumatoid Arthritis".
3. The dissertation should be based on personal observation and experience of the writer.
4. The competition is open to graduates of any Australian university.
5. The trustees reserve the right to withhold the award.
6. Essays must be delivered to the Medical Secretary, British Medical Association (Victorian Branch), by 4 p.m. on December 31, 1950.
7. Each essay must be typewritten or printed and must not exceed 75,000 words in length.
8. Each essay must be distinguished by a motto and must be accompanied by a sealed envelope marked by the same motto, containing the name and address of the author.
9. The trustees reserve the right to publish the prize essay.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales, as duly qualified medical practitioners:

Brown, James Alexander Spence, M.R.C.S. (England), L.R.C.P. (London), 1925, B.Ch. (Cantab.), 1926, Jonson Street, Byron Bay.

Gallent, John, M.B., B.S., 1948 (Univ. Melbourne), 27 Heygarth Street, Echuca, Victoria.

McCarthy, John Joseph, M.B., B.S., 1947 (Univ. Melbourne), St. Margaret's Hospital, Sydney.

Rowlands, Eustace Alwynne, M.B., B.S., 1930 (Univ. Melbourne), F.R.C.S. (England), 1934, F.R.A.C.S., 1938, 120 Collins Street, Melbourne.

Seith, Wolfram Immanuel, M.B., B.S., 1949 (Univ. Adelaide), District Hospital, Broken Hill.

Simmonds, Wilfred John, M.B., B.S., 1942 (Univ. Queensland), Kanematsu Institute, Sydney Hospital, Sydney.

Wells, William James Crosbie, M.B., Ch.B., 1929 (Univ. New Zealand), Oceanmore Hotel, Bondi.

Wellsted, Edward James, M.B., B.S., 1949 (Univ. Melbourne), 18 Gallipoli Street, Lidcombe.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED MAY 20, 1950.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.	Australia.[3]
Ankylostomiasis	*	..	..	..	..	..	..	..	..
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	4(1)	1(1)	..	..	..	1	..	..	6
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(*a*)	*	*	..	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	2(1)	..	..	..	..	..	2
Diphtheria	7(1)	15(5)	7(4)	1	3(2)	1	..	..	34
Dysentery (Amœbic)	*	2(2)	..	..	..	..	..	..	2
Dysentery (Bacillary)	*	1(1)	5(5)	..	1	..	..	..	7
Encephalitis Lethargica	..	..	..	..	..	..	..	..	..
Erysipelas	*	*	*	..	*	*	..	..	..
Filariasis	*	..	..	..	..	..	..	..	..
Helminthiasis	*	..	*	*	*	*	*	*	..
Hydatid	*	1	*	*	..	*	*	..	1
Influenza	*	*	*	..	*	*	..	*	..
Lead Poisoning	*	*	..	*	*	*	*	*	..
Leprosy	..	..	..	..	..	..	..	..	..
Malaria(*b*)	*	..	..	..	..	..	..	..	..
Measles	*	*	*	12(11)	*	*	..	3	15
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	12(7)	4(2)	..	26(19)	4(3)	..	..	..	46
Psittacosis	*	..	*	..	*	*	*	*	..
Puerperal Fever	..	..	1	..	..	..	..	..	1
Rubella(*c*)	*	*	..	*	..	*	..	..	..
Scarlet Fever	17(12)	29(21)	6(4)	6(2)	4(4)	3	..	..	65
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	..	2(2)	*	*	*	*	*	2
Trachoma	*	..	*	*	*	*	*	*	..
Tuberculosis(*d*)	37(24)	15(9)	4(2)	7(7)	8(6)	3(1)	..	..	74
Typhoid Fever(*e*)	..	..	..	..	..	..	..	..	..
Typhus (Endemic)(*f*)	..	..	..	..	1(1)	..	..	..	1
Undulant Fever	..	..	..	..	..	..	..	..	..
Weil's Disease(*g*)	*	*	*	*	*	*	*	*	..
Whooping Cough	*	*	*	6(6)	*	*	..	..	6
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 37, 1946-1947. Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from the Northern Territory.
* Not notifiable.
(*a*) Includes Mossman and Sarina fevers. (*b*) Mainly relapses among servicemen infected overseas. (*c*) Notifiable disease in Queensland in females aged over fourteen years. (*d*) Includes all forms. (*e*) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (*f*) Includes scrub, murine and tick typhus. (*g*) Includes leptospiroses, Weil's and para-Weil's disease.

The following additional qualifications have been registered:

Jacobs, Lionel Allenby, Kirribilli Avenue, Kirribilli (M.B., Ch.B., 1942, Univ. New Zealand), F.R.C.S. (Edinburgh), 1948, F.R.C.S. (England), 1949.

McGlynn, Ronald William, 7 Orinoco Road, Pymble (M.B., B.S., 1943, Univ. Sydney), M.Ch. (Orth.) (Univ. Liverpool), 1949.

Royle, Norman James, Pittwater Road, Pymble (M.B., B.S., 1940, Univ. Sydney), M.Ch. (Orth.) (Univ. Liverpool), 1949.

Watts, Archibald William James, 53 McIntosh Street, Gordon (M.B., B.S., 1943, Univ. Sydney), M.Ch. (Orth.) (Univ. Liverpool), 1949.

TASMANIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

McCluskie, John Aloysius, M.B., Ch.B., 1926, B.Sc., 1925 (Univ. Glasgow), Millbrook Rise, New Norfolk.

Woodward, William Winslow, M.B., 1946 (Univ. Sydney), Launceston General Hospital, Launceston.

Bierman, Basil Nathan, M.B., B.S., 1948 (Univ. Sydney), Launceston General Hospital, Launceston.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts*, 1939 *to* 1948, of Queensland, as duly qualified medical practitioners:

Skinn, Alfred John, M.B., Ch.B., 1925 (Univ. Edinburgh), c.o. Travel Bureau, Bank of New South Wales, Brisbane.

Henley, Maire, M.B., B.Ch., B.A.O., 1941 (National Univ. Ireland), c.o. Hospitals Board, Mackay.

The following additional qualifications have been registered:

Salkeld, Ormond William, Bennett Street, Toowong, Brisbane, D.O.M.S., R.C.P. and S. (London), 1949.

Thomson, Evan Rees Whitaker, 9 Fortitude Street, Auchenflower, Brisbane, F.R.C.S. (England), 1949.

University Intelligence.

UNIVERSITY OF MELBOURNE.

THE sixteenth Beattie-Smith Memorial Lectures on Insanity will be given this year by Professor O. A. Oeser, M.A., M.Sc., Ph.D., F.B.Ps.S., Professor of Psychology in the University of Melbourne. His general subject will be "The Conditions of Civilized Living". The first lecture will be given on Monday, June 19, 1950, the subject being "The Fields of Psychiatry and Psychology"; the chairman will be Professor R. D. Wright. The second lecture will be given on Monday, June 26, 1950, the subject being "Psychology and Preventive Medicine"; the chairman will be Dr. J. K. Adey. The lectures will be given in the Anatomy Theatre, University of Melbourne, at 8.15 p.m.

Medical Appointments.

Dr. Keith Archer has been appointed Government Medical Officer at Winton, New South Wales.

Dr. K. M. Bowden has been appointed Senior Government Pathologist of Victoria.

Dr. Patricia R. Davey has been appointed honorary assistant ear, nose and throat surgeon at the Royal Alexandra Hospital for Children, Camperdown.

Dr. B. E. Frecker has been appointed honorary assistant radiologist at the Royal Alexandra Hospital for Children, Camperdown.

Dr. R. J. Fleming and Dr. E. W. Bate have been appointed Government Pathologists of Victoria.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Guymer, Max William, M.B., B.S., 1947 (Univ. Adelaide), 28 Glasgow Avenue, Bondi.

Diary for the Month.

JUNE 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

JUNE 15.—South Australian Branch, B.M.A.: Special General Meeting.

JUNE 19.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.

JUNE 20.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135 Macquarie Street, Sydney): Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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